

EXHIBIT 33

Electroconvulsive therapy

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Electroconvulsive therapy (ECT), also known as **electroshock**, is a well-established, albeit controversial, psychiatric treatment in which seizures are electrically induced in anesthetized patients for therapeutic effect. Today, ECT is most often used as a treatment for severe major depression which has not responded to other treatment,^[1] and is also used in the treatment of mania (often in bipolar disorder), catatonia and schizophrenia. It was first introduced in the 1930s^[2] and gained widespread use as a form of treatment in the 1940s and 1950s; today, an estimated 1 million people worldwide receive ECT every year,^[3] usually in a course of 6–12 treatments administered 2 or 3 times a week.

Electroconvulsive therapy can differ in its application in three ways: electrode placement, length of time that the stimulus is given, and the property of the stimulus. The variance of these three forms of application have significant differences in both adverse side effects and positive outcomes. After treatment, drug therapy can be continued, and some patients receive continuation/maintenance ECT.

Informed consent is a standard of modern electroconvulsive therapy.^[4] Involuntary treatment is uncommon in countries that follow contemporary standards and is typically only used when the use of ECT is believed to be potentially life saving.^[5]

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Indications

There is considerable variability in opinion among experts as to whether ECT is appropriate as a first-line treatment or if its use should be reserved for patients who have not responded to other interventions such as medication and psychotherapy.^[6]

The APA 2001 guidelines give the primary indications for ECT among patients with depression as a lack of a response to, or intolerance of, antidepressant medications; a good response to previous ECT; and the need for a rapid and definitive response (e.g. because of psychosis or a risk of suicide). The decision to use ECT depends on several factors, including the severity and chronicity of the depression, the likelihood that alternative treatments would be effective, the patient's preference, and a weighing of the risks and benefits.^[7]

Some guidelines recommend that cognitive behavioral therapy or other psychotherapy should generally be tried before ECT is used. However, treatment resistance is widely defined as lack of therapeutic response to two antidepressants. The APA states that at times patients will prefer to receive ECT over alternative treatments, but commonly the opposite will be the case.

The APA ECT guidelines state that severe major depression with psychotic features, manic delirium, or catatonia are conditions for which there is a clear consensus favoring early reliance on ECT. The NICE guidelines recommend ECT for patients with severe depression, catatonia, or prolonged or severe mania.^[8]

The 2001 APA guidelines support the use of ECT for relapse prevention, but the 2003 NICE guidelines do not.

The 2001 APA ECT guidelines say that ECT is rarely used as a first-line treatment for schizophrenia but is considered after unsuccessful treatment with antipsychotic medication, and may also be considered in the treatment of patients with schizoaffective or schizophreniform disorder. The 2003 NICE ECT guidelines do not recommend ECT for Schizophrenia.

The NICE 2003 guidelines state that doctors should be particularly cautious when considering ECT treatment for women who are pregnant and for older or younger people, because they may be at higher risk of complications with ECT. The 2001 APA ECT guidelines say that ECT may be safer than alternative treatments in the infirm elderly and during pregnancy, and the 2000 APA depression guidelines stated that the literature supports the safety for mother and fetus, as well as the efficacy during pregnancy.

Non-clinical patient characteristics

About 70 percent of ECT patients are women.^[9] This is largely, but not entirely, due to the fact that women are more likely to receive treatment for depression.^{[9][10]} Older and more affluent patients are also more likely to receive ECT. The use of ECT treatment is "markedly reduced for ethnic minorities."^{[10][11]}

Effectiveness

The 1999 U.S. Surgeon General's Report on Mental Health summarized psychiatric opinion at the time about the effectiveness of ECT. It stated that both clinical experience and published studies had determined ECT to be effective (with an average 60 to 70 percent remission rate) in the treatment of severe depression, some acute psychotic states, and mania. Its effectiveness had not been demonstrated in dysthymia, substance abuse, anxiety, or personality disorder. The report stated that ECT does not have a long-term protective effect against suicide and should be regarded as a short-term treatment for an acute episode of illness, to be followed by continuation therapy in the form of drug treatment or further ECT at weekly to monthly intervals.^[12] A 2004 large multicentre clinical follow-up study of ECT patients in New York—describing itself as the first systematic documentation of the effectiveness of ECT in community practice in the 65 years of its use—found remission rates of only 30–47 percent, with 64 percent of those relapsing within six months.^[13]

ECT on its own does not usually have a sustained benefit. Virtually all those who remit end up relapsing within six months following a course, even when given a placebo.^[14] The relapse rate in the first six months may be reduced by the use of psychiatric medications or further ECT, but remains high.^{[15][16]}

Most, but not all, published reviews of the literature have concluded that ECT is effective in the treatment of depression. In 2006, research psychiatrist Colin A. Ross reviewed the entire body of placebo-controlled literature on ECT and found that no study demonstrated a significant difference between real and placebo ECT at one month post-treatment. The review also found that many of these studies failed to find a difference between real and placebo ECT even during the period of treatment. Based on these observations, Dr. Ross concludes that "claims in textbooks and review articles that ECT is effective are not consistent with the published data", and that consent forms for the procedure should state that "real ECT is only marginally more effective than placebo." The review was highly critical of other published reviews concluding that ECT was effective, because these reviews often relied primarily on studies that were not placebo-controlled.^[17]

Adverse effects

Aside from effects in the brain, the general physical risks of ECT are similar to those of brief general anesthesia; the United States' Surgeon General's report says that there are "no absolute health contraindications" to its use.^[12] Immediately following treatment the most common adverse effects are confusion and memory loss. The state of confusion usually disappears after a few hours.^{[18][19]}

Effects on memory

It is the effects of ECT on long-term memory that give rise to much of the concern surrounding its use.^[20] The acute effects of ECT can include amnesia, both retrograde (for events occurring before the treatment) and anterograde (for events occurring after the treatment).^[21] Memory loss and confusion are more pronounced with bilateral electrode placement rather than unilateral, and with sine-wave rather than brief-pulse currents. The vast majority of modern treatment uses brief pulse currents.^[21] Research by Harold Sackeim has shown that excessive current causes more risk for memory loss, and shocking only the right side of the head protects the left side, which contains the brain's verbal structure.^[22]

Retrograde amnesia is most marked for events occurring in the weeks or months before treatment, with one study showing that although some people lose memories from years prior to treatment, recovery of

such memories was "virtually complete" by seven months post-treatment, with the only enduring loss being memories in the weeks and months prior to the treatment.^{[23][24]} Anterograde memory loss is usually limited to the time of treatment itself or shortly afterwards. In the weeks and months following ECT these memory problems gradually improve, but some people have persistent losses, especially with bilateral ECT.^{[9][21]} One published review summarized the results of seven studies reporting on perceived memory loss and found that between 29% and 55% of respondents believed they experienced long-lasting or permanent memory changes.^[25] In 2000, American psychiatrist Sarah Lisanby and colleagues found that bilateral ECT left patients with persistent impairment for memory of public events as compared to RUL ECT.^[20]

Studies have found that patients are often unaware of substantial cognitive deficits induced by ECT.^[26]^[27] For example, in June, 2008, a Duke University study^[26] was published assessing the neuropsychological effects and attitudes in patients after ECT. Forty-six patients participated in the study, which involved neuropsychological and psychological testing before and after ECT. The study documented substantial cognitive decline after ECT on a variety of memory tests, including "verbal memory for word lists and prose passages and visual memory of geometric designs." The study further found that a significant number of patients erroneously believed that their memory had improved after ECT despite the fact that neuropsychological testing clearly showed the opposite. As stated by the researchers, "Indeed, there was a slight trend towards [patients reporting] improved memory functioning, despite the objective neuropsychological data indicating significantly lower recognition and delayed recall." Based on their findings, the authors issued the following recommendation:

"When ECT is provided to adolescents, the potential impact of such cognitive changes should be discussed with the patients and their parents or guardians in terms of implications for not only the patient's emotional functioning but cognitive functioning as well, particularly upon his or her academic performance. In summary, we argue that an individual cost-benefit analysis should be made in light of the implications of the potential benefits versus costs of ECT upon improving emotional functioning and the impact that potential memory changes may have on real-world functioning and quality of life."^[26]

Controversy over long-term effects on general cognition

According to prominent ECT researcher Harold Sackeim, "despite over fifty years of clinical use and ongoing controversy", until 2007 there had "never been a large-scale, prospective study of the cognitive effects of ECT."^[28] In this first-ever large-scale study (347 subjects), Sackeim and colleagues found that at least some forms (namely bilateral application and sine wave currents) of ECT "routine[ly]" lead to "adverse cognitive effects," including global cognitive deficits and memory loss, that persist for at least six months after treatment, suggesting that the induced deficits may be permanent.^{[28][29]} The authors also warned that their findings did not suggest that right-unilateral ECT did not also lead to chronic cognitive deficits.

Harold Sackeim can be seen in a videotaped deposition briefly discussing the findings of this study and why, in his opinion, earlier studies had failed to find evidence of long-term harm from ECT.^[30] Despite over fifty years of clinical use, Sackeim states that prior to 2001, "the field itself never really had an opportunity to have a discussion about patients who have complaints about long-term memory loss." In this video clip, Sackeim also reveals that at a California ECT conference with 200 practitioners present, when polled as to whether they think ECT can lead to chronic cognitive deficits, two-thirds raised their hands. Sackeim says this was "almost a watershed moment for the field", and was the "first time *publicly* that the field itself said 'no' to the position that it can't happen."^{[30][31]}

In July, 2007, a second study was published concluding that ECT routinely leads to chronic, substantial

cognitive deficits, and the findings were not limited to any particular forms of ECT.^[32] The study, led by psychiatrist Glenda MacQueen and colleagues, found that patients treated with ECT for bipolar disorder show marked deficits across multiple cognitive domains. According to the researchers, "Subjects who had received remote ECT had further impairment on a variety of learning and memory tests when compared with patients with no past ECT. This degree of impairment could not be accounted for by illness state at the time of assessment or by differential past illness burden between patient groups." Despite the findings of chronic, global cognitive deficits in post-ECT patients, MacQueen and colleagues suggest that it is "unlikely that such findings, even if confirmed, would significantly change the risk-benefit ratio of this notably effective treatment."^[32]

Six months after the publication of the Sackeim study^[28] documenting routine, long-term memory loss after ECT, prominent ECT researcher Max Fink published a review in the journal *Psychosomatics* concluding that patient complaints of memory loss after ECT are "rare" and should be "characterized as somatoform disorders, rather than as evidence of brain damage, thus warranting psychological treatment for such disorders."^[33] Based on his findings, Fink suggests that, "Instead of endorsing these reports as the direct consequence of ECT, especially in patients who have recovered from their depressive illness, lost their suicidal drive, and have improved social functioning, is it not more useful to accept the complaint as a somatoform disorder, explore the basis in the individual's history and experience, and offer appropriate supportive treatment?"^[33]

Most recent reviews of the literature and other articles continue to characterize ECT as safe and effective.^{[34][35][36][37][38][39][40][41]} For example, in June, 2009, Portuguese researchers published a review on the safety and efficacy of ECT in an article entitled, *Electroconvulsive Therapy: Myths and Evidences*.^[34] In their review, the researchers conclude that ECT is an "efficient, safe and even life saving treatment for several psychiatric disorders." In 2008, Yale researchers published a review on the safety and efficacy of ECT in elderly patients.^[41] According to the authors, "ECT is well established as a safe and effective treatment for several psychiatric disorders." And in a June, 2009, article published in the *Journal of ECT*, Iranian researchers observe that, "Despite the wide consensus over the safety and efficacy of electroconvulsive therapy (ECT), it still faces negative publicity and unfavorable attitudes of patients and families."^[40]

Psychiatrist Peter Breggin, chief editor of the journal *Ethical Human Psychology and Psychiatry*, is a leading critic of ECT who believes the procedure is neither safe nor effective. In a published article reviewing the findings of Harold Sackeim's 2007 study^[28] on the cognitive effects of ECT, Breggin accuses Max Fink and other pro-ECT researchers of having a history of "systematically covering up damage done to millions of [ECT] patients throughout the world."^[29] He disagrees with the position that findings of chronic, global cognitive deficits should have no bearing on the risk-benefit ratio of ECT, and he believes it's important to address the "actual impact of these losses on the lives of individual patients." In a section of his paper entitled *Destroying Lives*, Dr. Breggin writes, "Even when these injured people can continue to function on a superficial social basis, they nonetheless suffer devastation of their identities due to the obliteration of key aspects of their personal lives. The loss of the ability to retain and learn new material is not only humiliating and depressing but also disabling. Even when relatively subtle, these activities can disrupt routine activities of living."^[29]

A study published in 2004 in the *Journal of Mental Health* reported that 35 to 42% of patients said ECT resulted in loss of intelligence.^[42] The study also reported, "There is no overlap between clinical and consumer studies on the question of benefit."

A recent article by a neuropsychologist and a psychiatrist in Dublin suggests that ECT patients who

experience cognitive problems following ECT should be offered some form of cognitive rehabilitation. The authors say that the failure to attempt to rehabilitate patients may be partly responsible for the negative public image of ECT.^[43]

Effects on brain structure

Considerable controversy exists over the effects of ECT on brain tissue despite the fact that a number of mental health associations, including the American Psychiatric Association, have concluded that there is no evidence that ECT causes structural brain damage.^{[8][44]} A 1999 report by the United States Surgeon General states, "The fears that ECT causes gross structural brain pathology have not been supported by decades of methodologically sound research in both humans and animals".^[5] However, not all experts agree that ECT does not cause brain damage, and two studies have been published since 2007 finding that at least some forms of ECT may result in *widespread, persisting, generalized cognitive dysfunction*, which would seem to support claims that ECT causes brain damage.^{[28][32][45]}

A leading critic of ECT, psychiatrist Peter Breggin has published books and reviews of the literature purporting to show that ECT routinely causes brain damage as evidenced by a considerable list of studies in humans and animals.^[46] In particular, Dr. Breggin asserts that animal and human autopsy studies have shown that ECT routinely causes '*widespread pinpoint hemorrhages and scattered cell death*.'^[45] According to Dr. Breggin, the 1990 APA task force report on ECT ignored much of the scientific literature pointing out the negative effects of electroshock therapy. For example, in 1952 Hans Hartelius conducted and published an animal study on cats entitled *Cerebral Changes Following Electrically Induced Convulsions* in which a double-blind microscopic pathology examination showed that it was possible to distinguish the 8 shocked animals from the 8 non-shocked animals with remarkable accuracy based on statistically significant structural changes to the brain, including vessel wall changes, gliosis, and nerve cell changes. Based on the detection of shadow cells and neuronophagia, Hartelius determined that there was irreversible damage to neurons associated with electroshock.^[45]

Proponents argue that the addition of hyperoxygenation and refinement in technique in the last thirty years has made ECT safe, and a majority of published reviews in recent decades have reflected this position.^[47] In a 2004 study designed to evaluate whether modern ECT techniques lead to identifiable brain damage, twelve monkeys underwent daily electroshock for six weeks under conditions meant to simulate human ECT; the animals were then sacrificed and their brains were compared to monkeys undergoing anesthesia alone. According to the researchers, "None of the ECT-treated monkeys showed pathological findings."^[48]

There are recent animal studies that have documented significant brain damage after an electroshock series. For example, in 2005, Russian researchers published a study entitled, *Electroconvulsive Shock Induces Neuron Death in the Mouse Hippocampus: Correlation of Neurodegeneration with Convulsive Activity*. In this study, the researchers found that after an electroshock series, there was a significant loss of neurons in parts of the brain and particularly in defined parts of the hippocampus where up to 10% of neurons were killed. The researchers conclude that "the main cause of neuron death is convulsions evoked by electric shocks."^[49] In 2008, Portuguese researchers conducted a rat study aimed at answering the question of whether an electroshock series causes structural changes in vulnerable parts of the brain.^[50] According to the authors, "This study answers positively the question of whether repeated administration of ECS seizures can cause brain lesions. Our data are consistent with findings from other animal models and from human studies in showing that neurons located in the entorhinal cortex and in the hilus of the dentate gyrus are particularly vulnerable to repeated seizures."

Many expert proponents of ECT maintain that the procedure is safe and does not cause brain damage. Dr. Charles Kellner, a prominent ECT researcher and former chief editor of the *Journal of ECT* states in a recent published interview that, "There are a number of well-designed studies that show ECT does not cause brain damage and numerous reports of patients who have received a large number of treatments over their lifetime and have suffered no significant problems due to ECT."^[51] Dr. Kellner cites specifically to a study purporting to show an absence of cognitive impairment in eight subjects after more than 100 lifetime ECT treatments.^[52] One of the authors of the cited study, Harold Sackeim, published a large-scale study less than a month after this interview concluding that the type of ECT used in the eight patients receiving the 100 lifetime treatments, bilateral sine wave, routinely leads to persistent, global cognitive deficits^[28] (discussed supra). Dr. Kellner states that, "Rather than cause brain damage, there is evidence that ECT may reverse some of the damaging effects of serious psychiatric illness."^[51]

Effects in pregnancy

If steps are taken to decrease potential risks, ECT is generally accepted to be relatively safe during all trimesters of pregnancy, particularly when compared to pharmacological treatments.^{[53][54][55]} Suggested preparation for ECT during pregnancy includes a pelvic examination, discontinuation of nonessential anticholinergic medication, uterine tocodynamometry, intravenous hydration, and administration of a nonparticulate antacid. During ECT, elevation of the pregnant woman's right hip, external fetal cardiac monitoring, intubation, and avoidance of excessive hyperventilation are recommended.^[53] Much of the medical literature in this area is composed of case studies of single or twin pregnancies, and although some have reported serious complications,^{[56][57]} the majority have found ECT to be safe.^[58]

Administration

Informed consent is sought before treatment. Patients are informed about the risks and benefits of the procedure. Patients are also made aware of risks and benefits of other treatments and of not having the procedure done at all. Depending on the jurisdiction the need for further inputs from other medical professionals or legal professionals may be required. ECT is usually given on an in-patient basis. Prior to treatment a patient is given a short-acting anesthetic such as methohexital, propofol, etomidate, or thiopental,^[9] a muscle relaxant such as suxamethonium (succinylcholine), and occasionally atropine to inhibit salivation.

Both electrodes can be placed one on the same side of the patient's head. This is known as unilateral ECT. Unilateral ECT is used first to minimize side effects (memory loss). When electrodes are placed on both sides of the head, this is known as bilateral ECT. In bifrontal ECT, an uncommon variation, the electrode position is somewhere between bilateral and unilateral. Unilateral is thought to cause fewer cognitive effects than bilateral but is considered less effective.^[9] In the USA most patients receive bilateral ECT.^[59] In the UK almost all patients receive bilateral ECT.^[60]

The electrodes deliver an electrical stimulus. The stimulus levels recommended for ECT are in excess of an individual's seizure threshold: about one and a half times seizure threshold for bilateral ECT and up to 12 times for unilateral ECT.^[9] Below these levels treatment may not be effective in spite of a seizure, while doses massively above threshold level, especially with bilateral ECT, expose patients to the risk of more severe cognitive impairment without additional therapeutic gains.^[61] Seizure threshold is determined by trial and error ("dose titration"). Some psychiatrists use dose titration, some still use "fixed dose" (that is, all patients are given the same dose) and others compromise by roughly estimating a

patient's threshold according to age and sex.^[59] Older men tend to have higher thresholds than younger women, but it is not a hard and fast rule, and other factors, for example drugs, affect seizure threshold.

ECT machines

Most modern ECT machines deliver a brief-pulse current, which is thought to cause fewer cognitive effects than the sine-wave currents which were originally used in ECT.^[9] A small minority of psychiatrists in the USA still use sine-wave stimuli.^[59] Sine-wave is no longer used in the UK.^[60] Typically, the electrical stimulus used in ECT is about 800 milliamps and has up to several hundred watts, and the current flows for between one and 6 seconds.^[61] In the USA, ECT machines are manufactured by two companies, Somatics, which is owned by psychiatrists Richard Abrams and Conrad Swartz, and Mecta. The Food and Drug Administration has classified the devices used to administer ECT as Class III medical devices.^[62] Class III is the highest-risk class of medical devices. In the UK, the market for ECT machines was long monopolized by Ectron Ltd, although in recent years some hospitals have started using American machines. Ectron Ltd was set up by psychiatrist Robert Russell, who together with a colleague from the Three Counties Asylum, Bedfordshire, invented the Page–Russell technique of intensive ECT.

Variations in international practice

There is wide variation in ECT use between different countries, different hospitals, and different psychiatrists.^[9] International practice varies considerably from widespread use of the therapy in many western countries to a small minority of countries that do not use ECT at all, such as Slovenia.^[63] Guidelines on the use of ECT are stringent in the USA and the UK. Modern standards are not always followed throughout the world and not all countries that use ECT have written technical standards. The use of both anesthesia and muscle relaxants is universally recommended in the administration of ECT. If anesthesia and muscle relaxants are not used the procedure is called unmodified ECT. In a minority of countries such as Japan,^[64] India,^[65] and Nigeria,^[66] ECT may be used without anesthesia. WHO has called for a worldwide ban on unmodified ECT and the topic is currently being debated in countries like India. The practice has been recently abolished in Turkey's largest psychiatric hospital.^[67] A major difficulty for developing countries in eliminating unmodified ECT is a lack of trained anesthesiologists available to administer the procedure.^[68] A small minority of countries never seek consent before administering ECT. This significantly uneven application of ECT around the world continues to make ECT a controversial procedure.

Sarah Hall reports, "ECT has been dogged by conflict between psychiatrists who swear by it, and some patients and families of patients who say that their lives have been ruined by it. It is controversial in some European countries such as the Netherlands and Italy, where its use is severely restricted".^[69]

United States

In the USA, a survey of psychiatric practice in the late 1980s found that an estimated 100,000 people received ECT annually, with wide variation between metropolitan statistical areas.^[70] Accurate statistics about the frequency, context and circumstances of ECT in the United States are difficult to obtain because only a few states have reporting laws that require the treating facility to supply state authorities with this information.^[71] One state which does report such data is Texas, where in the mid-1990s ECT was used in about one third of psychiatric facilities and given to about 1,650 people annually.^[10] Usage of ECT has since declined slightly; in 2000–01 ECT was given to about 1,500 people aged from 16 to 97

(in Texas it is illegal to give ECT to anyone under sixteen).^[72] ECT is more commonly used in private psychiatric hospitals than in public hospitals, and minority patients are underrepresented in the ECT statistics.^[9] In the United States, ECT is usually given three times a week; in the UK, it is usually given twice a week.^[9] Occasionally it is given on a daily basis.^[9] A course usually consists of 6–12 treatments, but may be more or fewer. Following a course of ECT some patients may be given continuation or maintenance ECT with further treatments at weekly, fortnightly or monthly intervals.^[9] A few psychiatrists in the USA use multiple-monitored ECT (MMECT) where patients receive more than one treatment per anesthetic.^[9] As of 2007, electroconvulsive therapy practice is not officially regulated in the USA, and the education of physicians in its prescription and administration has been described as poor. Electroconvulsive therapy is not a required subject in US medical schools and not a required skill in psychiatric residency training. Privileging for ECT practice at institutions is a local option, no national certification standards are established, and no ECT-specific continuing training experiences are required of ECT practitioners.^[73]

United Kingdom

In the United Kingdom in 1980, an estimated 50,000 people received ECT annually, with use declining steadily since then^{[74][75]} to about 12,000 per annum. It is still used in nearly all psychiatric hospitals, with a survey of ECT use from 2002 finding that 71 percent of patients were women and 46 percent were over 65 years of age. Eighty-one percent had a diagnosis of mood disorder; schizophrenia was the next most common diagnosis. Sixteen percent were treated without their consent.^[76] In 2003, the National Institute for Clinical Excellence, a government body which was set up to standardize treatment throughout the National Health Service, issued guidance on the use of ECT. Its use was recommended "only to achieve rapid and short-term improvement of severe symptoms after an adequate trial of treatment options has proven ineffective and/or when the condition is considered to be potentially life-threatening in individuals with severe depressive illness, catatonia or a prolonged manic episode".^[77] The guidance received a mixed reception. It was welcomed by an editorial in the British Medical Journal^[78] but the Royal College of Psychiatrists launched an unsuccessful appeal.^[79] The NICE guidance, as the British Medical Journal editorial points out, is only a policy statement and psychiatrists may deviate from it if they see fit. Adherence to standards has not been universal in the past. A survey of ECT use in 1980 found that more than half of ECT clinics failed to meet minimum standards set by the Royal College of Psychiatrists, with a later survey in 1998 finding that minimum standards were largely adhered to, but that two-thirds of clinics still fell short of current guidelines, particularly in the training and supervision of junior doctors involved in the procedure.^[80] A voluntary accreditation scheme, ECTAS, was set up in 2004 by the Royal College, but as of 2006 only a minority of ECT clinics in England, Wales, Northern Ireland and the Republic of Ireland have signed up.^[81]

Mechanism of action

The aim of ECT is to induce a therapeutic clonic seizure (a seizure where the person loses consciousness and has convulsions) lasting for at least 15 seconds. Although a large amount of research has been carried out, the exact mechanism of action of ECT remains elusive. The main reasons for this are the difficulty of isolating the therapeutic effect from the plethora of effects that accompany the anesthetic, electric shock and seizure; the differences between the brains of humans and those of other animals; and the lack of satisfactory animal models of mental illness.^[9]

Electroconvulsive Therapy (ECT) increases serum brain-derived neurotrophic factor (BDNF) in drug resistant depressed patients.^[82]

Legal status

Informed consent

It is widely acknowledged internationally that obtaining the written, informed consent of the patient is important before ECT is administered. The World Health Organization, in its 2005 publication "Human Rights and Legislation WHO Resource Book on Mental Health," specifically states, "ECT should be administered only after obtaining informed consent."^[83]

In the US, this doctrine places a legal obligation on a doctor to make a patient aware of: the reason for treatment, the risks and benefits of a proposed treatment, the risks and benefits of alternative treatment, and the risks and benefits of receiving no treatment. The patient is then given the opportunity to accept or reject the treatment. The form states how many treatments are recommended and also makes the patient aware that the treatment may be revoked at anytime during a course of ECT.^[12] The Surgeon General's Report on Mental Health states that patients should be warned that the benefits of ECT are short-lived without active continuation treatment in the form of drugs or further ECT, and that there may be some risk of permanent, severe memory loss after ECT.^[12] The report advises psychiatrists to involve patients in discussion, possibly with the aid of leaflets or videos, both before and during a course of ECT.

To demonstrate what he believes should be required to fully satisfy the legal obligation for informed consent, one psychiatrist, working for an anti-psychiatry organisation, has formulated his own consent form^[84] using the consent form developed and enacted by the Texas Legislature as a model.^[85]

In the UK, in order for consent to be valid it requires an explanation in "broad terms" of the nature of the procedure and its likely effects.^[86] One review from 2005 found that only about half of patients felt they were given sufficient information about ECT and its adverse effects,^[87] and another survey found that about fifty percent of psychiatrists and nurses agreed with them.^[88]

A 2005 study published in the *British Journal of Psychiatry* described patients' perspectives on the adequacy of informed consent before ECT.^[89] The study found that, "About half (45–55%) of patients reported they were given an adequate explanation of ECT, implying a similar percentage felt they were not." The authors also stated:

"Approximately a third did not feel they had freely consented to ECT even when they had signed a consent form. The proportion who feel they did not freely choose the treatment has actually increased over time. The same themes arise whether the patient had received treatment a year ago or 30 years ago. Neither current nor proposed safeguards for patients are sufficient to ensure informed consent with respect to ECT, at least in England and Wales."^[89]

Involuntary ECT

Procedures for involuntary ECT vary from country to country depending on local mental health laws. Legal proceedings are required in some countries, while in others ECT is seen as another form of treatment that may be given involuntarily as long as legal conditions are observed.

In most states in the USA, a judicial order following a formal hearing is needed before a patient can be forced to undergo involuntary ECT. Patients may be represented by legal counsel at the hearing. Oregon Revised Statutes allow for involuntary ECT with the signature of a physician independent of the patient's facility, and no judicial order or legal counsel are required. According to the Surgeon General's Report on

Mental Health, "As a rule, the law requires that such petitions are granted only where the prompt institution of ECT is regarded as potentially lifesaving, as in the case of a person in grave danger because of lack of food or fluid intake caused by catatonia."^[12] However, there are legal loopholes that thwart strict adherence to this principle. For example, an American citizen was being forced to undergo ECT against his will in 2009, even though his life was not in danger.^{[90][91]} In this March 17, 2009 video, the man, his mother, and advocates, speak out against his forced ECT. The description of the video states that "Though Sandford, 54, is not charged with any crime, he has received over 40 such rounds of shocks on an outpatient basis so far – even after his original mental problems have long since subsided and he has repeatedly asked for the shocks to stop. Over the objections of Sandford, his mother and friends, his legal conservator at Lutheran Social Service of MN (LSSMN) has gone to court and succeeded in mandating a continuation of the procedure." The TCIMC reports that "Like all other USA states, Minnesota has [legal] loopholes allowing [its] citizens to receive electroshock over their expressed wishes."^[92]

In England and Wales the Mental Health Act 1983 currently allows the use of ECT on detained patients (with and without capacity) if the treatment is likely to alleviate or prevent deterioration in a condition and is authorized by a psychiatrist from the Mental Health Act Commission's panel. However, proposed amendments to the Mental Health Act (clause 30) will introduce a capacity-threshold for the imposition of ECT. This in effect will mean that ECT may not be given to a patient who has capacity to refuse to consent to it, irrespective of his or her detention under the Act (the treatment may still be given in an emergency under s62).^[93] If the treating psychiatrist thinks the need for treatment is urgent they may start a course of ECT before authorization.^[94] About 2,000 people a year in England and Wales are treated without their consent under the Mental Health Act,^[95] with a small number of informal patients treated in this way under common law. In Scotland the Mental Health (Care and Treatment) (Scotland) Act 2003 gives patients with capacity the right to refuse ECT.^[96]

Duress in involuntary ECT makes reports about its effects, by patients while under duress, uncertain in their validity. Megara Sanderson, Events & Culture Editor of newspaper "The Flying Horse" reports, "Some speculate that patients may pretend that they are cured of their mental illness so that they no longer have to endure the electro-convulsive treatment."^{[97][98]}

Involuntary electroshock contravenes the principle of autonomy in medical ethics. The maxim of autonomy is "Voluntas aegroti suprema lex." This rule states that the will of the patient is supreme. It implies that a patient has the right to refuse a medical treatment, such as ECT.

History

As early as the 16th century, agents to produce seizures were used to treat psychiatric conditions. In 1785, the therapeutic use of seizure induction was documented in the London Medical Journal.^[9] Convulsive therapy was introduced in 1934 by Hungarian neuropsychiatrist Ladislas J. Meduna who, believing mistakenly that schizophrenia and epilepsy were antagonistic disorders, induced seizures with first camphor and then metrazol (cardiazol).^[99] Within three years metrazol convulsive therapy was being used worldwide.^[100] In 1937, the first international meeting on convulsive therapy was held in Switzerland by the Swiss psychiatrist Muller. The proceedings were published in the American Journal of Psychiatry and, within three years, cardiazol convulsive therapy was being used worldwide.^[100] Italian Professor of neuropsychiatry Ugo Cerletti, who had been using electric shocks to produce seizures in animal experiments, and his colleague Lucio Bini developed the idea of using electricity as a substitute for metrazol in convulsive therapy and, in 1937, experimented for the first time on a person. Sherwin B. Nuland, having discussed the matter with a first-hand observer in the 1970s, gave the following description of the results of the first use of ECT on a person:

"They thought, 'Well, we'll try 55 volts, two-tenths of a second. That's not going to do anything terrible to him.' So they did that. [...] This fellow — remember, he wasn't even put to sleep — after this major grand mal convulsion, sat right up, looked at these three fellows and said, 'What the fuck are you assholes trying to do?' Well, they were happy as could be, because he hadn't said a rational word in the weeks of observation."^[101]

ECT soon replaced metrazol therapy all over the world because it was cheaper, less frightening and more convenient.^[102] Cerletti and Bini were nominated for a Nobel Prize but did not receive one. By 1940, the procedure was introduced to both England and the US. Through the 40's and 50's the use of ECT became widespread. ECT is the only form of shock treatment still performed by modern medicine.

In the early 1940s, in an attempt to reduce the memory disturbance and confusion associated with treatment, two modifications were introduced: the use of unilateral electrode placement and the replacement of sinusoidal current with brief pulse. It took many years for brief-pulse equipment to be widely adopted.^[103] Unilateral ECT has never been popular with psychiatrists and is still only given to a minority of ECT patients.^[9] In the 1940s and early 1950s ECT was usually given in "unmodified" form, without muscle relaxants, and the seizure resulted in a full-scale convulsion. A rare but serious complication of unmodified ECT was fracture or dislocation of the long bones. In the 1940s psychiatrists began to experiment with curare, the muscle-paralysing South American poison, in order to modify the convulsions. The introduction of suxamethonium (succinylcholine), a safer synthetic alternative to curare, in 1951 led to the more widespread use of "modified" ECT. A short-acting anesthetic was usually given in addition to the muscle relaxant in order to spare patients the terrifying feeling of suffocation that can be experienced with muscle relaxants.^[103]

The steady growth of antidepressant use along with negative depictions of ECT in the mass media led to a marked decline in the use of ECT during the 50's to the 70's. The Surgeon General stated there were problems with electroshock therapy in the initial years before anesthesia was routinely given and, *these now antiquated practices contributed to the negative portrayal of ECT in the popular media.*^[104] The New York Times described the public's negative perception of ECT as being caused mainly by one movie, "For Big Nurse in *One Flew Over the Cuckoo's Nest*, it was a tool of terror, and in the public mind *shock therapy* has retained the tarnished image given it by Ken Kesey's novel: dangerous, inhumane and overused".^[22]

In 1976, Dr. Blatchley demonstrated the effectiveness of his constant current, brief pulse device ECT. This device eventually largely replaced earlier devices because of the reduction in cognitive side effects, although some ECT clinics in the US still use sine-wave devices.^[59] The 1970s saw the publication of the first American Psychiatric Association task force report on electroconvulsive therapy (to be followed by further reports in 1990 and 2001). The report endorsed the use of ECT in the treatment of depression. The decade also saw criticism of ECT.^[105] Specifically critics pointed to shortcomings such as noted side effects, the procedure being used as a form of abuse, and uneven application of ECT. The use of ECT declined until the 1980s, "when use began to increase amid growing awareness of its benefits and cost-effectiveness for treating severe depression".^[104] In 1985 the National Institute of Mental Health and National Institutes of Health convened a consensus development conference on ECT and concluded that, whilst ECT was the most controversial treatment in psychiatry and had significant side-effects, it had been shown to be effective for a narrow range of severe psychiatric disorders.^[106]

Due to the backlash noted previously, national institutions reviewed past practices and set new standards. In 1978, The American Psychiatric Association released its first task force report in which new standards for consent were introduced and the use of unilateral electrode placement was recommended. The 1985

NIMH Consensus Conference confirmed the therapeutic role of ECT in certain circumstances. The American Psychiatric Association released its second task force report in 1990 where specific details on the delivery, education, and training of ECT were documented. Finally in 2001 the American Psychiatric Association released its latest task force report. This report emphasizes the importance of informed consent, and the expanded role that the procedure has in modern medicine.

Patient experience

The APA ECT taskforce guidelines report findings that most patients find ECT no worse than going to the dentist, and many found it less stressful than the dentist. They report that other research finds that most patients would voluntarily receive ECT again if needed.

NICE ECT guidelines report that some individuals consider ECT to have been a beneficial and lifesaving treatment, while others reported feelings of terror, shame and distress, and found it positively harmful and an abusive invasion of personal autonomy, especially when administered without their consent.

Individual positive depictions

Kitty Dukakis, wife of politician Michael Dukakis, reports in a *Newsweek* article mostly positive effects from electroconvulsive therapy, and regards memory loss as an acceptable price to pay for relief from depression.

For me, the memory issues are real but manageable. Things I lose generally come back. Other memories I prefer to lose, including those about the depression I was suffering. But there are some memories—of meetings I have attended, people's homes I have visited—that I don't want to lose but I can't help it. They generally involve things I did two weeks before and two weeks after ECT. Often they are just wiped out....I have learned ways to partly compensate for whatever loss I still experience. I call my sister Jinny, Michael and my kids, asking what my niece Betsy's phone number is, what we did yesterday and what we are planning to do tomorrow. I apologize prior to asking. I wonder when they are going to run out of patience with "Kitty being Kitty." I hate losing memories, which means losing control over my past and my mind, but the control ECT gives me over my disabling depression is worth this relatively minor cost. It just is.^[107]

American psychotherapist Martha Manning's autobiographical *Undercurrents*^[108] acknowledges the downside of treatment: "I felt like I'd been hit by a truck for a while, but that was, comparatively speaking, not so bad," as well as the upside: "Afterwards, I thought, do regular people feel this way all the time? It's like you've not been in on a great joke for the whole of your life."

In his autobiographical book *Electroboy*, American writer Andy Behrman describes undergoing ECT as a treatment for bipolar disorder while under house-arrest: "I wake up thirty minutes later and think I am in a hotel in Acapulco. My head feels as if I have just downed a frozen margarita too quickly. My jaws and limbs ache. But I am elated."^[109]

Curtis Hartmann, a lawyer in western Massachusetts, stated: "ECT, a treatment of last resort for severe, debilitating depression, is all that has ever worked for me. I awaken about 20 minutes later, and although I am still groggy with anesthesia, much of the hellish depression is gone. It is a disease that for me, literally steals me from myself—a disease that executes me and then forces me to stand and look down at my corpse. Thankfully, ECT has kept my monster at bay, my hope intact".^[110]

Individual negative depictions

Negative effects of ECT have been reported by noteworthy individuals.

Ernest Hemingway, American author, committed suicide shortly after ECT at the Menninger Clinic in 1961. He is reported to have said to his biographer, "Well, what is the sense of ruining my head and erasing my memory, which is my capital, and putting me out of business? It was a brilliant cure but we lost the patient..."^[111]

In 2005, "Peggy S. Salters, 60, sued Palmetto Baptist Medical Center in Columbia, as well as the three doctors responsible for her care. As the result of an intensive course of outpatient ECT in 2000, she lost all memories of the past 30 years of her life, including all memories of her husband of three decades, now deceased, and the births of her three children. Ms. Salters held a Masters of Science in nursing and had a long career as a psychiatric nurse, but lost her knowledge of nursing skills and was unable to return to work after ECT."^[112] The jury awarded Salters \$635,177 in compensation for her inability to work.

Registered nurse Barbara C. Cody reports in a letter to the *Washington Post* that her life was forever changed by 13 outpatient ECTs she received in 1983. "Shock 'therapy' totally and permanently disabled me. EEGs [electroencephalograms] verify the extensive damage shock did to my brain. Fifteen to 20 years of my life were simply erased; only small bits and pieces have returned. I was also left with short-term memory impairment and serious cognitive deficits. [deletion] Shock 'therapy' took my past, my college education, my musical abilities, even the knowledge that my children were, in fact, my children. I call ECT a rape of the soul."^[113]

In 2007, a judge canceled a two year old court order that allowed the involuntary electroshock of Simone D., a psychiatric patient at Creedmoor Psychiatric Center in the state of New York.^[114] Although Simone spoke only Spanish, she rarely received access to staff fluent in her language.^{[114][115]} Simone previously had 200 electroshocks.^{[114][115]} However, she communicated that she did not want more electroshock.^{[114][115]} Simone stated, "Electroshock causes more pain. I suffer more from shock treatment!"^[114] "

In 2008, David Tarloff, a psychiatric patient who had received electroshock, assaulted two therapists in the city of New York. Tarloff injured one therapist and murdered the other. One of the therapists was Kent Shinbach, a psychiatrist who had an interest in electroconvulsive therapy. "It is not clear whether Dr. Shinbach played any role in Mr. Tarloff's shock therapy".^[116] However, Tarloff told investigators that Shinbach had given Tarloff psychiatric treatment at a psychiatric facility initially in 1991.^[117]

In an interview with *Houston Chronicle* in 1996, Melissa Holliday, a former extra on *Baywatch* and model for *Playboy* stated the ECT she received in 1995, "ruined her life." She went on to state, "I've been through a rape, and electroshock therapy is worse. If you haven't gone through it, I can't explain it."^[118]

Liz Spikol, the senior contributing editor of *Philadelphia Weekly*, wrote of her ECT in 1996, "Not only was the ECT ineffective, it was incredibly damaging to my cognitive functioning and memory. But sometimes it's hard to be sure of yourself when everyone "credible"—scientists, ECT docs, researchers—are telling you that your reality isn't real. How many times have I been told my memory loss wasn't due to ECT but to depression? How many times have I been told that, like a lot of other consumers, I must be perceiving this incorrectly? How many times have people told me that my feelings of trauma related to the ECT are misplaced and unusual? It's as if I was raped and people kept telling me not to be upset—that it wasn't that bad."^[119]

Public perception and mass media

A questionnaire survey of 379 members of the general public in Australia indicated that more than 60% of respondents had some knowledge about the main aspects of ECT. Participants were generally opposed to the use of ECT on depressed individuals with psychosocial issues, on children, and on involuntary patients. Public perceptions of ECT were found to be mainly negative.^[120]

Fictional and semi-fictional depictions

Main article: Fictional and semi-fictional depictions of ECT

Electroconvulsive therapy has been depicted in several fictional, non-fictional and semi-fictional films, books, television shows, stage musicals, and songs, such as in *Frances*, *Requiem for a Dream*, *One Flew Over the Cuckoo's Nest*, *Il Meglio Gioventu' (The Best of Youth)*, *House*, *The Bell Jar*; *Girl, Interrupted*, *Insanitarium*, *Changeling*, *Next to Normal*, and *Return to Oz*

Famous people who have undergone ECT

- Caroline Aherne, British comedian writer and actress, most commonly known for portraying 'Denise' in 'The Royle Family'
- Antonin Artaud, French poet and playwright.^[121]
- Dick Cavett, American television talk show host.^[122]
- Paulo Coelho, author of *The Alchemist*.^[123]
- Kitty Dukakis, wife of former Massachusetts governor and 1988 Democratic presidential nominee Michael Dukakis and author of *Shock*,^[124] a book chronicling her experiences with ECT.^[107]
- Thomas Eagleton, US senator and vice presidential candidate.^[125]
- Roky Erickson, American singer, songwriter, harmonica player and guitarist.^[126]
- Carrie Fisher, American actress and novelist.^[127] Fisher speaks at length of her experiences with ECT in her autobiography *Wishful Drinking*.
- Janet Frame, New Zealand writer and poet.^[128]
- Judy Garland, American actress.^[129]
- Harold Gimblett, British cricketer.^[130]
- Peter Green, English blues guitarist, founding member of Fleetwood Mac.^[131]
- Roy Harper, English musician.
- David Helfgott, Australian pianist.^[132]
- Ernest Hemingway, American Pulitzer Prize winning novelist, Nobel Laureate, short-story writer, and journalist.^[133]
- Marya Hornbacher, American writer.^[134]
- Vladimir Horowitz, Russian-American classical pianist. ^[135]
- Vivien Leigh, English actress and second wife of Laurence Olivier.^[136]
- Michael Moriarty, American actor.^[137]
- Sherwin B. Nuland, American surgeon and writer.^[138]
- Robert M. Pirsig, American author of *Zen and the Art of Motorcycle Maintenance*.^[139]
- Sylvia Plath, American writer and poet.^[140]
- Bud Powell, American jazz musician.^[141]
- Lou Reed, American singer-songwriter.^[142]
- Yves Saint-Laurent, French fashion designer.^[143]

- Edie Sedgwick, American socialite and Warhol Superstar.^[144]
- Gene Tierney, American actress.^[145]
- Townes van Zandt, American country singer-songwriter.^[146]
- David Foster Wallace, American writer.^[147]

Lou Reed- band member of The Velvet Underground and self made famous musician and guitarist.

Wikimedians who have undergone ECT

- Nevit Dilmen

See also

- DSM-IV Codes
- Harold A. Sackeim
- Insulin shock therapy

References

1. ^ Scott AIF (ed) *et al.* (2005). "The ECT Handbook Second Edition: The Third Report of the Royal College of Psychiatrists' Special Committee on ECT" (PDF). Royal College of Psychiatrists. <http://www.rcpsych.ac.uk/files/pdfversion/cr128.pdf>. Retrieved 2008-07-26.
2. ^ Psychology Frontiers and Applications – Second Canadian Edition (Passer, Smith, Atkinson, Mitchell, Muir)
3. ^ "Electroconvulsive therapy discussion hosted at the MGH". <http://www2.massgeneral.org/pubaffairs/issues2006/101306ect.htm>. Retrieved 2007-06-05.
4. ^ Carla Curran (2007-09-17). "Shock Therapy Makes a Comeback: States Respond". Ncs1.org. <http://www.ncsl.org/programs/health/shn/2007/sn499c.htm>. Retrieved 2009-10-17.
5. ^ ^a ^b Mental Health: A Report of the Surgeon General – Chapter 4. Retrieved 2007-12-29.
6. ^ Task Force on Electroconvulsive Therapy. The practice of electroconvulsive therapy: recommendations for treatment, training, and privileging. 2nd ed. Washington, DC: American Psychiatric Publishing, 2001.
7. ^ Lisanby, S.H. (2007) Electroconvulsive Therapy for Depression Volume 357, No. 19, pp. 1939–1945
8. ^ ^a ^b "Guidance on the use of electroconvulsive therapy" (PDF). National Institute for Clinical Excellence. 2005-11-01. <http://www.nice.org.uk/nicemedia/pdf/59ectfullguidance.pdf>. Retrieved 2008-07-26.
9. ^ ^a ^b ^c ^d ^e ^f ^g ^h ⁱ ^j ^k ^l ^m ⁿ ^o ^p Rudorfer, MV, Henry, ME, Sackeim, HA (2003). "Electroconvulsive therapy". In A Tasman, J Kay, JA Lieberman (eds) *Psychiatry, Second Edition*. Chichester: John Wiley & Sons Ltd, 1865–1901.
10. ^ ^a ^b ^c Reid WH, Keller S, Leatherman M, Mason M (January 1998). "ECT in Texas: 19 months of mandatory reporting". *J Clin Psychiatry* **59** (1): 8–13. PMID 9491059.
11. ^ Euba R, Saiz A (2006). "A comparison of the ethnic distribution in the depressed inpatient population and in the electroconvulsive therapy clinic". *J ECT* **22** (4): 235–6. doi:10.1097/01.yct.0000235928.39279.52. PMID 17143151.
12. ^ ^a ^b ^c ^d ^e Surgeon General (1999). *Mental Health: A Report of the Surgeon General*, chapter 4.
13. ^ Prudic J, Olfson M, Marcus SC, Fuller RB, Sackeim HA (2004). "Effectiveness of electroconvulsive therapy in community settings". *Biol. Psychiatry* **55** (3): 301–12. doi:10.1016/j.biopsych.2003.09.015. PMID 14744473.
14. ^ Sackeim HA, Haskett RF, Mulsant BH, Thase ME, Mann JJ, Pettinati HM, Greenberg RM, Crowe RR, Cooper TB, Prudic J.(2001) Continuation pharmacotherapy in the prevention of relapse following electroconvulsive therapy: a randomized controlled trial. *JAMA*. 2001 Mar 14; **285**(10):1299–307.
15. ^ Tew JD Jr, Mulsant BH, Haskett RF, Joan P, Begley AE, Sackeim HA. (2007) Relapse during continuation pharmacotherapy after acute response to ECT: a comparison of usual care versus protocolized treatment] *Ann*

- Clin Psychiatry. 2007 Jan–Mar; **19**(1):1–4 PMID 17453654
16. ^ Kellner CH, Knapp RG, Petrides G, Rummans TA, Husain MM, Rasmussen K, Mueller M, Bernstein HJ, O'Connor K, Smith G, Biggs M, Bailine SH, Malur C, Yim E, McClintock S, Sampson S, Fink M. (2006) Continuation electroconvulsive therapy vs pharmacotherapy for relapse prevention in major depression: a multisite study from the Consortium for Research in Electroconvulsive Therapy (CORE). *Arch Gen Psychiatry*. 2006 Dec; **63**(12):1337–44. PMID 17146008
 17. ^ Ross CA (2006). "The sham ECT literature: implications for consent to ECT.". *Ethical Human Psychiatry and Psychology* **8** (1): 17–28. doi:10.1891/ehpp.8.1.17. PMID 16856307.
 18. ^ "Electroshock". DepNet. <http://www.depnet.co.in/universe1/treatment/ect/>. Retrieved 2009-03-18.
 19. ^ "Electroconvulsive therapy – Electroshock (ECT)". DoctorsLounge. <http://www.doctorslounge.com/psychiatry/procedures/ect.htm>. Retrieved 2009-03-19.
 20. ^ ^{a b} Lisanby SH, Maddox JH, Prudic J, Devanand DP, Sackeim HA (June 2000). "The effects of electroconvulsive therapy on memory of autobiographical and public events". *Arch. Gen. Psychiatry* **57** (6): 581–90. doi:10.1001/archpsyc.57.6.581. PMID 10839336. <http://archpsyc.ama-assn.org/cgi/pmidlookup?view=long&pmid=10839336>.
 21. ^ ^{a b c} Benbow, SM (2004) "Adverse effects of ECT". In AIF Scott (ed.) *The ECT Handbook, second edition*. London: The Royal College of Psychiatrists, pp. 170–174.
 22. ^ ^{a b} Goleman, Daniel (1990-08-02). "The Quiet Comeback of Electroshock Therapy". *The New York Times*: p. B5. <http://query.nytimes.com/gst/fullpage.html?res=9C0CE0D81F3EF931A3575BC0A966958260&pagewanted=all>. Retrieved 2008-01-01.
 23. ^ Squire LR, Slater PC, Miller PL (January 1981). "Retrograde amnesia and bilateral electroconvulsive therapy. Long-term follow-up". *Arch. Gen. Psychiatry* **38** (1): 89–95. PMID 7458573.
 24. ^ Squire LR, Slater PC (January 1983). "Electroconvulsive therapy and complaints of memory dysfunction: a prospective three-year follow-up study". *Br J Psychiatry* **142**: 1–8. doi:10.1192/bjp.142.1.1. PMID 6831121.
 25. ^ Rose, D *et al.* (2003). "Patients' perspectives on electroconvulsive therapy: systematic review". *British Medical Journal* **326**:1363–1365.
 26. ^ ^{a b c} Feliu M, *et al.* (June 2008). "Neuropsychological effects and attitudes in patients following electroconvulsive therapy.". *Neuropsychiatric Disease and Treatment* **4** (3): 613–17. PMID 18830401. PMC 2526376. <http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pubmed&pubmedid=18830401>.
 27. ^ Coleman EA, Sackeim HA, *et al.* (March 1996). "Subjective Memory Complaints Prior to and Following Electroconvulsive Therapy.". *Biological Psychiatry*. **39** (5): 346–56. doi:10.1016/0006-3223(95)00185-9. PMID 18830401.
 28. ^ ^{a b c d e f} Sackeim HA, Prudic J, Fuller R, Keilp J, Lavori PW, Olfson M (January 2007). "The cognitive effects of electroconvulsive therapy in community settings". *Neuropsychopharmacology* **32** (1): 244–54. doi:10.1038/sj.npp.1301180. PMID 16936712.
 29. ^ ^{a b c} Breggin Peter (2007). "ECT Damages the Brain: Disturbing News for Patients and Shock Doctors Alike.". *Ethical Human Psychology and Psychiatry*. **9** (2): 83–86. doi:10.1891/152315007782021196. http://breggin.com/index2.php?option=com_docman&task=doc_view&gid=203&Itemid=3.
 30. ^ ^{a b} "The Awakening; Harold Sackeim (You Tube)". 2004. <http://www.youtube.com/watch?v=hvwVRn91bAU>. Retrieved September 22, 2009.
 31. ^ "All Clips from Harold Sackeim Deposition (You Tube)". 2004. http://www.youtube.com/results?search_query=harold+sackeim&search_type=&aq=f. Retrieved September 22, 2009.
 32. ^ ^{a b c} MacQueen G., *et al.* (2007). "The long-term impact of treatment with electroconvulsive therapy on discrete memory systems in patients with bipolar disorder". *Journal of Psychiatry and Neuroscience* **32** (4): 241–249. PMID 17653292. PMC 1911194. <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1911194>.
 33. ^ ^{a b} Fink, M. (July–August 2007). "Complaints of loss of personal memories after electroconvulsive therapy: evidence of a somatoform disorder?". *Psychosomatics* **48** (4): 290–93. doi:10.1176/appi.psy.48.4.290. PMID 17600164.
 34. ^ ^{a b} Coentre R, *et al.* (May–June 2009). "Electroconvulsive therapy: myths and evidences.". *Acta Medica Portuguesa* **22** (3): 275–80. PMID 19686628. <http://www.actamedicaportuguesa.com/pdf/2009-22/3/275-280.pdf>.
 35. ^ Zyss T, *et al.* (Nov–Dec 2008). "Shock therapy in psychiatry--historical feature.". *Psychiatria Polska* **42** (6): 797–818. PMID 19441660.
 36. ^ Faedda GL, *et al.* (March 2009). "The origins of electroconvulsive therapy: Prof. Bini's first report on

- ECT". *Journal of affective disorders*. **Epub Ahead of Print**. doi:10.1016/j.jad.2009.01.023. PMID 19268370.
37. ^ van Herck E, et al. (2009). "Electroconvulsive therapy for patients with intracranial aneurysms: a case study and literature review". *Dutch Journal of Psychiatry* **51** (1): 43–51.
http://www.tijdschriftvoorpsychiatrie.nl/en/tijdschrift/artikel/TVPart_2736.
 38. ^ Cristancho MA, et al. (Dec 2008). "Uncommon but serious complications associated with electroconvulsive therapy: recognition and management for the clinician". *Current psychiatry reports* **10** (6): 474–80. doi:10.1007/s11920-008-0076-4. PMID 18980730.
 39. ^ Busnello JV, et al. (Dec 2008). "Peripheral nucleotide hydrolysis in rats submitted to a model of electroconvulsive therapy.". *Prog Neuropsychopharmacol Biol Psychiatry*. **32** (8): 1829–33. doi:10.1016/j.pnpbp.2008.08.007. PMID 18775464.
 40. ^ ^{a b} Malekian A, et al. (Jun 2009). "Knowledge of attitude toward experience and satisfaction with electroconvulsive therapy in a sample of Iranian patients.". *Journal of ECT*. **25** (2): 106–12. doi:10.1097/YCT.0b013e31818050dc. PMID 18708944.
 41. ^ ^{a b} Wilkins KM, et al. (March 2008). "Efficacy of electroconvulsive therapy in the treatment of nondepressed psychiatric illness in elderly patients: a review of the literature.". *Journal of geriatric psychiatry and neurology*. **21** (1): 3–11. doi:10.1177/0891988707311027. PMID 18287164.
 42. ^ Philpot M, Collins C, Trivedi P, Treloar A, Gallacher S, Rose D: Eliciting users' views of ECT in two mental health trusts with a user-designed questionnaire. *Journal of Mental Health* **13**(4): 403–413, 2004
 43. ^ Mangaoang, MA and Lacey, JV (2007). "Cognitive rehabilitation: assessment and treatment of persistent memory impairments following ECT". *Advances in Psychiatry* **13**: 90–100.
 44. ^ American Psychiatric Association. "Electroconvulsive Therapy (ECT)".
http://www.psych.org/Departments/APIREandResearch/ResearchTraining/clin_res/index.aspx. Retrieved 2007-12-29.
 45. ^ ^{a b c} Breggin, Peter R. (2007). *Brain Disabling Treatments in Psychiatry: Drugs, Electroshock, and the Psychopharmaceutical Complex*. New York: Springer Publishing Company. pp. 237–239. ISBN 082612934X. <http://books.google.com/books?id=hBd0V7Ex8PUC&lpg=PA237&ots=IpegPI-NSJ&dq=ECT%20study%20brain%20damage&pg=PA237#v=onepage&q=ECT%20study%20brain%20damage&f=false>.
 46. ^ Breggin, Peter (1981). "Disabling the Brain with Electroshock". http://breggin.com/index.php?option=com_docman&task=doc_download&gid=167&Itemid=37.
 47. ^ Folkerts H (2000). "Electroconvulsive therapy of depressive disorders.". *Therapeutische Umschau* **57** (2): 90–94. PMID 10730103.
 48. ^ Dwork AJ, et al. (March 2004). "Absence of histological lesions in primate models of ECT and magnetic seizure therapy.". *The American Journal of Psychiatry* **161** (3): 576–78. doi:10.1176/appi.ajp.161.3.576. PMID 14992989.
 49. ^ Zarubenko II, et al. (september 2005). "Electroconvulsive shock induces neuron death in the mouse hippocampus: correlation of neurodegeneration with convulsive activity.". *Neuroscience and Behavioral Physiology* **35** (7): 715–721. doi:10.1007/s11055-005-0115-0. PMID 16433067.
 50. ^ Cardoso A, et al. (January 2008). "Loss of synapses in the entorhinal-dentate gyrus pathway following repeated induction of electroshock seizures in the rat.". *Journal of Neuroscience Research* **86** (1): 71–83. doi:10.1002/jnr.21474. PMID 17705293.
 51. ^ ^{a b} "In Session with Charles H. Kellner, MD: Current Developments in Electroconvulsive Therapy, Interviewed by Dr. Norman Sussman, December, 2006". [Primarypsychiatry.com](http://www.primarypsychiatry.com).
<http://www.primarypsychiatry.com/asp/articleDetail.aspx?articleid=1028>. Retrieved 2009-10-17.
 52. ^ Devanand DP, Sackeim HA, et al. (july 1991). "Absence of cognitive impairment after more than 100 lifetime ECT treatments.". *The American Journal of Psychiatry* **148** (7): 929–32. PMID 2053635.
 53. ^ ^{a b} Miller LJ (May 1994). "Use of electroconvulsive therapy during pregnancy". *Hosp Community Psychiatry* **45** (5): 444–50. PMID 8045538. <http://ps.psychiatryonline.org/cgi/pmidlookup?view=long&pmid=8045538>.
 54. ^ Walker R, Swartz CM (September 1994). "Electroconvulsive therapy during high-risk pregnancy". *Gen Hosp Psychiatry* **16** (5): 348–53. doi:10.1016/0163-8343(94)90022-1. PMID 7995506.
 55. ^ Ferrill MJ, Kehoe WA, Jacisin JJ (1992). "ECT During Pregnancy: Physiologic and Pharmacologic Considerations". *Convuls Ther* **8** (3): 186–200. PMID 11941169.
 56. ^ e.g. PMID 10668602 (death) (French), PMID 10074880 (premature labour), PMID 9871846 (spontaneous abortion)

57. ^ Pinette MG, Santarpio C, Wax JR, Blackstone J (August 2007). "Electroconvulsive therapy in pregnancy". *Obstet Gynecol* **110** (2 Pt 2): 465–6. doi:10.1097/01.AOG.0000265588.79929.98 (inactive 2008-09-08). PMID 17666629.
58. ^ e.g. PMID 14560648, PMID 17804997, PMID 11593722, PMID 10614034 (two patients), PMID 8198651 (twins), PMID 11941159 (twins), PMID 2246991
59. ^ ^{a b c d} Prudic J, Olfson M, Sackeim HA (July 2001). "Electro-convulsive therapy practices in the community". *Psychol Med* **31** (5): 929–34. doi:10.1017/S0033291701003750. PMID 11459391.
60. ^ ^{a b} Duffett R, Lelliott P (1998). "Auditing electroconvulsive therapy. The third cycle". *Br J Psychiatry* **172**: 401–5. doi:10.1192/bjp.172.5.401. PMID 9747401.
61. ^ ^{a b} Lock, T (1995). "Stimulus dosing". In C Freeman (ed.) *The ECT Handbook*. London: Royal College of Psychiatrists, 72–87.
62. ^ Federal Register (1979), p. 51776
63. ^ See the Slovenian government website for information about ECT in Slovenia.
64. ^ Motohashi N, Awata S, Higuchi T (2004). "A questionnaire survey of ECT practice in university hospitals and national hospitals in Japan". *J ECT* **20** (1): 21–3. doi:10.1097/00124509-200403000-00005. PMID 15087992.
65. ^ Chanpattana W, Kunigiri G, Kramer BA, Gangadhar BN (2005). "Survey of the practice of electroconvulsive therapy in teaching hospitals in India". *J ECT* **21** (2): 100–4. doi:10.1097/01.yct.0000166634.73555.e6. PMID 15905751.
66. ^ Ikeji OC, Ohaeri JU, Osahon RO, Agidee RO (1999). "Naturalistic comparative study of outcome and cognitive effects of unmodified electro-convulsive therapy in schizophrenia, mania and severe depression in Nigeria". *East Afr Med J* **76** (11): 644–50. PMID 10734527.
67. ^ "Abusive practice of "unmodified" electroshock treatment abolished at main psychiatric facility of Turkey". Disabled Peoples' International. http://v1.dpi.org/lang-en/resources/topics_detail?page=557. Retrieved 2008-03-25.
68. ^ Dutta, Rita (2003). "Psychiatrists plead against ban of direct electro convulsive therapy". Indian Express Group of Newspapers. <http://www.expresshealthcaremgmt.com/20030531/medethics.shtml>. Retrieved 2007-12-31.
69. ^ Rise In Electric Shock Therapy In County. Sarah Hall, Norwich Evening News 24, June 4, 2008. Accessed: June 4, 2008.
70. ^ Hermann R, Dorwart R, Hoover C, Brody J (1995). "Variation in ECT use in the United States". *Am J Psychiatry* **152** (6): 869–75. PMID 7755116.
71. ^ Cauchon, Dennis (1995-12-06). "Patients often aren't informed of full danger". USA Today.
72. ^ Texas Department of State (2002) Electroconvulsive therapy reports.
73. ^ Fink, M. & Taylor, A.M. (2007) Electroconvulsive therapy: Evidence and Challenges JAMA Vol. 298 No. 3, p330–332.
74. ^ Pippard, J and Ellam, L (1981). *Electroconvulsive treatment in Great Britain, 1980*. London: Gaskell.
75. ^ Pippard J, Ellam L (1981). "Electroconvulsion treatment in Great Britain 1980". *Lancet* **2** (8256): 1160–1. PMID 6118592.
76. ^ Electro convulsive therapy: survey covering the period from January 2002 to March 2002, *Statistical Bulletin* 2003/08. Department of Health.
77. ^ NICE 2003. Electroconvulsive therapy (ECT). Retrieved on 2007-12-29.
78. ^ Carney, S & Geddes, J (2003). "Electroconvulsive therapy: recent recommendations are likely to improve standards and uniformity of use". *British Medical Journal* **326**: 1343–4.
79. ^ NICE (2003). Appraisal of electroconvulsive therapy: decision of the appeal panel. London: NICE.
80. ^ Duffett, R and Lelliott, P (1998). "Auditing electroconvulsive therapy: the third cycle". *British Journal of Psychiatry* **172**:401–405.
81. ^ Royal College of Psychiatrists (2006). ECTAS newsletter issue 5.
82. ^ Bocchio-Chiavetto L, Zanardini R, Bortolomasi M, *et al* (2006). "Electroconvulsive Therapy (ECT) increases serum Brain Derived Neurotrophic Factor (BDNF) in drug resistant depressed patients". *Eur Neuropsychopharmacol* **16** (8): 620–4. doi:10.1016/j.euroneuro.2006.04.010. PMID 16757154.
83. ^ World Health Organisation (2005). *WHO Resource Book on Mental Health, Human Rights and Legislation*. Geneva, 64.
84. ^ Johnson, R. "An informed consent form for electroconvulsive therapy, draft 1." (PDF). PsychRights. <http://www.psychrights.org/Research/Digest/InformedConsent/DrJohnsonECTInformedConsent.pdf>.
85. ^ Texas Legislature (2004). Health & Safety Code Chapter 578, Electroconvulsive And Other Therapies Sec.578.001.

86. ^ Jones, R (1996) *Mental Health Act Manual, 5th edition*. London: Sweet and Maxwell, page 225.
87. ^ Rose, DS *et al.* (2005) "Information, consent and perceived co-ercion: patients' perspectives on electroconvulsive therapy". *British Journal of Psychiatry* **186**:54–59.
88. ^ Lutchman, RD *et al.* (2001). "Mental health professionals' attitudes towards and knowledge of electroconvulsive therapy." *Journal of Mental Health* **10**(20):141–150.
89. ^ ^{a b} Rose D, Wykes T, Bindman J, Fleischmann P: Information, consent and perceived coercion: patients' perspectives on electroconvulsive therapy. *British Journal of Psychiatry* **186**: 54–59, 2005.
90. ^ Benson, Lorna (2008) "Man Fights Order To Undergo Electroshock Therapy" *National Public Radio, Day to Day, December 15, 2008* <http://www.npr.org/templates/story/story.php?storyId=98273451>
91. ^ "Campaign to End Forced Outpatient Electroshock of Ray Sandford" *Mindfreedom, Gateway: Campaign to End Forced Outpatient Electroshock of Ray Sandford* <http://www.mindfreedom.org/ray>
92. ^ Twin Cities Indymedia (2009) *Ray Sandford, Advocates Speak Out Against Forced Electroshocks* <http://www.tc.indymedia.org/2009/mar/their-own-words-ect-survivor-advocates-speak-out-against-continued-forced-electroshocks>
93. ^ The Mental Health Act 1983, Part 4, section 58.
94. ^ The Mental Health Act 1983, Part 4, section 62.
95. ^ The Mental Health Act Commission (2005) *In Place of Fear? eleventh biennial report, 2003–2005*, 236. The Stationery Office.
96. ^ The Mental Health (Care and Treatment) (Scotland) Act 2003, Part 16, sections 237–239.
97. ^ Author: Megara Sanderson. Web page: "A Shocking Experience." Web site: "The Flying Horse." Date: April 14, 2008. Institution: Middlesex Community College. Date of access: May 2, 2008. Web address: <http://theflyinghorse.net/wp/?p=101>.
98. ^ Author: Megara Sanderson. Article: "A Shocking Experience." Periodical: "The Flying Horse." Volume: 2. Issue: 4. Date: May 2008. Institution: Middlesex Community College.
99. ^ Berrios G E (1997) The scientific origins of electroconvulsive therapy. *History of Psychiatry* **8**: 105-119.
100. ^ ^{a b} Fink, M (1984). "The origins of convulsive therapy". *American Journal of Psychiatry* **141**:1034–41.
101. ^ Nuland, S.: *Electroshock Therapy*. TED Talks, 2001, Monterey, CA, USA.
102. ^ Cerletti, U (1956). "Electroshock therapy". In AM Sackler *et al.* (eds) *The Great Physiodynamic Therapies in Psychiatry: an historical appraisal*. New York: Hoeber-Harper, 91–120.
103. ^ ^{a b} Kiloh, LG, Smith, JS, Johnson, GF (1988). *Physical Treatments in Psychiatry*. Melbourne: Blackwell Scientific Publications, 190–208. ISBN 0-86793-112-4
104. ^ ^{a b} Goode, Erica (1999-10-06). "Federal Report Praising Electroshock Stirs Uproar". *New York Times*. <http://query.nytimes.com/gst/fullpage.html?sec=health&res=9805E1DD1431F935A35753C1A96F958260>. Retrieved 2008-01-01.
105. ^ See Friedberg, J (1977). "Shock treatment, brain damage, and memory loss: a neurological perspective". *American Journal of Psychiatry* **134**:1010–1014; and Breggin, PR (1979) *Electroshock: its brain-disabling effects*. New York: Springer
106. ^ Blaine, JD and Clark, SM (1986). "Report of the NIMH–NIH consensus development conference on Electroconvulsive therapy". *Psychopharmacology Bulletin* **22**(2): 445–452.
107. ^ ^{a b} Dukakis, K and Tye, L (2006), excerpt from 'I Feel Good, I Feel Alive'. *Newsweek* September **18**:62–63.
108. ^ Manning, Martha (March 1996). *Undercurrents: A Therapist's Reckoning with Her Own Depression*. Harper San Francisco. ISBN 978-0062511843.
109. ^ Behrman, Andy (February 2003). *Electroboy: A Memoir of Mania*. Random House. ISBN 978-0812967081.
110. ^ "Personal Accounts: Life as Death: Hope Regained With ECT – Hartmann "'53'"(4): 413 – Psychiatr Serv". [Psychservices.psychiatryonline.org](http://psychservices.psychiatryonline.org). doi:10.1176/appi.ps.53.4.413. <http://psychservices.psychiatryonline.org/cgi/content/full/53/4/413>. Retrieved 2009-10-17.
111. ^ A. E. Hotchner, *Papa Hemingway: A Personal Memoir*, ISBN 0786705922; pg 280
112. ^ "ECT verdict awards dollar judgment". [Peoplewho.org](http://www.peoplewho.org). <http://www.peoplewho.org/documents/ctip.ectverdict.htm>. Retrieved 2009-10-17.
113. ^ Cody, Barbara (Approximately between 1995–2006). "Letter". [HealthyPlace.com](http://www.healthypace.com). <http://www.healthypace.com/Communities/Depression/ect/media/postltrs.html>. Retrieved 2006-09-23.
114. ^ ^{a b c d e} MindFreedom International. Another victory against forced electroshock. Simone D. wins! August 28, 2007. Accessed: April 18, 2008.
115. ^ ^{a b c} Lauren Tenney. Testimony from Lauren Tenney, Member of FUTURE Views and the Mental Patients Liberation Alliance. New York State Office of Mental Health, October 5, 2007. Accessed: April 18, 2008.
116. ^ "Shock Therapy Emerges As Detail in Therapist's Killing – February 21, 2008 – The New York Sun".

- Nysun.com. <http://www.nysun.com/new-york/shock-therapy-emerges-as-detail-in-therapists/71616/>. Retrieved 2009-10-17.
117. ^ Christopher Faherty (2008-04-02). "Suspected Killer 'Didn't Mean To Hurt Anyone'". <http://www.nysun.com/news/new-york/suspected-killer-didnt-mean-hurt-anyone>. Retrieved 2008-04-13.
 118. ^ "Woman Says Electric Shock Treatment Destroyed Her Life". [Healthplace.com](http://www.healthplace.com/Communities/Depression/ect/news/woman.asp). 1996-06-26. Retrieved 2009-10-17.
 119. ^ "Funday: ECT study shocks. Ha ha. | The Trouble With Spikol". [Trouble.philadelphiaweekly.com](http://trouble.philadelphiaweekly.com/archives/2006/12/funday_ect_stud.html). 2006-12-22. Retrieved 2009-10-17.
 120. ^ Teh, S.P.C., Helmes, E. & Drake, D. (2007) A Western Australian Survey On Public Attitudes Toward and Knowledge of Electroconvulsive Therapy *International Journal of Social Psychiatry*, Vol. 53, No. 3, 247–271 (2007) DOI: 10.1177/0020764006074522
 121. ^ Barber, Stephen (February 2005). *The Screaming Body: Antonin Artaud – Film Projects, Drawings and Sound Recordings*. Creation Books. ISBN 978-1840680911.
 122. ^ Abrams, Richard. *Electroconvulsive Therapy*, Oxford University Press, 2002
 123. ^ Coelho, Paulo (2006). *The Zahir*. HarperCollinsPublishers. ISBN 0007220855.
 124. ^ Dukakis, Kitty; Tye, Larry (September 2006). *Shock: The Healing Power of Electroconvulsive Therapy*. Avery. ISBN 978-1583332658.
 125. ^ "The White House losers". *Guardian*. 2008-03-29. <http://www.guardian.co.uk/politics/2008/mar/29/uselections2008.usa>. Retrieved 2008-03-29.
 126. ^ "Dallas – Music – Roky Erickson". [Dallasobserver.com](http://www.dallasobserver.com/2007-03-01/music/roky-erickson/). Retrieved 2009-10-17.
 127. ^ Fisher, Carrie (2008). *Wishful Drinking*. Simon & Schuster. p. 163. ISBN 978-1439102251.
 128. ^ Frame, J. *An Angel at My Table*, London, Virago, 2008 (autobiography)
 129. ^ Author: Citizens Commission on Human Rights. Web page: "Harming Artists: Psychiatry Ruins Creativity." Web site: "Psychiatric Abuse." Date: 2004. Institution: Citizens Commission on Human Rights. Date of access: May 28, 2008. Web address: <http://www.psychiatric-abuse.org.uk/Harming%20Artists.pdf>
 130. ^ "Tale of a tormented genius". <http://content-www.cricinfo.com/england/content/story/129485.html>. Retrieved 2007-06-12.
 131. ^ "Peter Green Biography". [Fmlegacy.com](http://www.fmlegacy.com/Bios/biopeter.html). Retrieved 2009-10-17.
 132. ^ "Denis Dutton on David Helfgott". [Denisdutton.com](http://www.denisdutton.com/helfgott.htm). Retrieved 2009-10-17.
 133. ^ "Ernest Hemingway (American writer) - Britannica Online Encyclopedia". [Britannica.com](http://www.britannica.com/EBchecked/topic/260825/Ernest-Hemingway.html). Retrieved 2009-10-17.
 134. ^ Hornbacher, Marya. *Madness: A Bipolar Life*, New York, 2008 (autobiography)
 135. ^ Plaskin, Glenn (1983). Biography of Vladimir Horowitz Quill ISBN 0-688-02656-7 Pages 338, 387, 389
 136. ^ Capua, Michelangelo (2003). *Vivien Leigh: A Biography*. McFarland. pp. 157, 169. ISBN 0-786-41497-9.
 137. ^ "Moriarty tames his demons". [Mmuuuhp.com](http://www.mmuuuhp.com/demons050720.htm). 2005-07-20. Retrieved 2009-10-17.
 138. ^ Sherwin Nuland (2001) My history of electroshock therapy, TED lecture
 139. ^ "Famous Shock Patients". [Ect.org](http://www.ect.org/famous-shock-patients/). Retrieved 2009-10-17.
 140. ^ "Sylvia Plath Homepage". [Sylviaplath.de](http://www.sylviaplath.de). Retrieved 2009-10-17.
 141. ^ Leland, John (2004). *Hip, the history*. HarperCollins. p. 123. ISBN 0-060-52817-6.
 142. ^ *Valley News* article: "Can electroshock therapy make you a better singer?"
 143. ^ "The Biography Channel – Yves Saint Laurent Biography". [Thebiographychannel.co.uk](http://www.thebiographychannel.co.uk/biography_story/931:1087/1/Yves_Saint_Laurent.htm). 2009-09-11. Retrieved 2009-10-17.
 144. ^ Stein, Jean; Plimpton, George (1994). *Edie: American Girl*. Grove Press. pp. 390, 398. ISBN 0-802-13410-6.
 145. ^ Shorter, Edward; Healy, David (2007). *Shock Therapy: A History of Electroconvulsive Treatment in Mental Illness*. Rutgers University Press. p. 156. ISBN 0-813-54169-7.
 146. ^ See the biographical film, *Be Here to Love Me*.
 147. ^ David Foster Wallace, Influential Writer, Dies at 46

External links

- American Psychiatric Association on ECT – brief article from the American Psychiatric Association.
- MIND on ECT – information on ECT from MIND (leading mental health charity in England and Wales).
- MindFreedom International - information critical of electroshock.
- About to have ECT? ... – Psychiatric Times article on the portrayal of ECT by Hollywood
- Electroconvulsive Therapy (ECT) – ECT products and devices from MECTA Corporation
- TED talk on ECT – 30-year-practicing surgeon and successful writer Sherwin B. Nuland on the history of ECT and his personal experience with it.

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Categories: Psychiatric treatments | Neurotechnology | Human rights abuses | History of mental health | Treatment of bipolar disorder

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EXHIBIT 34

CHAPTER 9

Electroconvulsive Therapy (ECT) for Depression

ECT is frequently used and retains enormous support within the medical profession. Despite recent scientific blows to their “treatment,” electroshock advocates remain determined, powerful, and influential. Anyone who doubts this need only read the September 12, 2007, issue of the *Journal of the American Medication Association (JAMA)* titled “Interest Surging in Electroconvulsive and Other Brain Stimulation Therapies” (Lamberg, 2007). Beneath a photo of health professionals hovering over an unconscious ECT patient, the caption reads, “Although studies have demonstrated that electroconvulsive therapy (ECT) is an effective and safe treatment for severe major depression, inaccurate perceptions of ECT contribute to lingering stigma and fear regarding its use.” This positive and even promotional attitude flies in the face of decades of research and heartrending patient testimonials. The publication of this puff piece at this time is probably intended to counter yet one more recently published scientific study that demonstrated the damaging effects of electroshocks to the brain (Sackeim et al., 2007).

Beginning in 1979 with the publication of my book *Electroshock: Its Brain-Disabling Effects*, followed by many other book chapters and scientific reports, I have marshaled innumerable studies, bolstered by my clinical experience, to show that electroconvulsive therapy (ECT) causes permanent brain dysfunction and damage, including widespread memory

and cognitive deficits. I have also evaluated evidence that contrary to claims that ECT prevents suicide, ECT is ineffective and actually causes or contributes to suicide.

Since the 1997 edition of this book, my task has been lightened by research from the heart of the ECT establishment confirming that ECT causes permanent brain damage and dysfunction with widespread cognitive deficits and that ECT greatly elevates the suicide risk, especially in the first week following treatment. In addition, a recent review of controlled clinical trials for ECT demonstrated once again that the so-called treatment is ineffective. And finally, for the first time in history, an ECT malpractice case has been won in court.

Since the ECT literature almost never provides clinical cases that describe the damage caused by the treatment, I will begin with a case from my own clinical practice.

A LIFE DESTROYED BY ECT

Sarah Williams was 55 years old when her husband died of a sudden heart attack in the early spring. She managed to teach music in high school for the remainder of the year, but by the summer, her "blues" worsened. She lost weight, had difficulty staying asleep at night, and even lost her zest for visiting with her grown children. Her oldest daughter, Jeannette, became concerned and in June took her to a psychiatrist. On the first visit, he put her on a tricyclic antidepressant, doxepin, that made her feel too groggy, so she stopped taking it. Then he put her on Prozac, which made her feel agitated. She was now both depressed and agitated, and her psychiatrist admitted her to a hospital for ECT.

Jeannette was very reluctant to submit her mother to ECT, but she was convinced by the doctor and a video film that shock was the most effective modality for depression. Jeannette and her mother were told that the electrical current and the grand mal convulsion that it produced were virtually harmless. The electrodes would be placed on only one side of the head (*unilateral ECT*), with the latest modifications to prevent injury.

Mrs. Williams herself protested about having electricity passed through her brain, and she wondered why no one seemed to want to talk with her about her feelings. Didn't psychiatrists do talking therapy anymore? But she was willing to accept anything that promised an end to the hopelessness that pervaded her life. She especially wanted to stop being a burden to her daughter Jeannette.

After the first shock treatment, Mrs. Williams developed a headache and stiff neck. She was somewhat nauseated. By the third treatment,

given every other day, she was confused and could not recall her daughter's previous visit. Her daughter was reassured by the doctor that this was "normal" for ECT, that all the effects were temporary, and that it would be best if she did not see her mother until the series of 10 ECTs was completed.

The nurse's notes from the hospitalization showed increasing "complaints" of memory difficulties by Mrs. Williams as the treatments progressed in number. However, after the eighth ECT, she stopped communicating about anything. The doctor's progress note at this point stated, "Improved. No longer complaining of feelings of depression." The nurse's progress note indicated, "No complaints. Sits quietly."

By the 10th treatment, Mrs. Williams could not find her way around the ward. The head of occupational therapy noted that the patient was too "disoriented and confused" to participate in the music and art activities.

When Jeannette visited her mother again at the conclusion of the treatments, she hardly recognized her. The expression on her mother's face was bland and indifferent, rather than pained. Sometimes her mother got a silly, almost goofy look that especially upset Jeannette. Her mother had always been so serious and dignified. To her daughter's dismay, her mother could not remember any of the events of the previous summer, including the visits to the psychiatrist. She could not remember who had come to her husband's funeral the previous April. She could not remember much about teaching for two semesters during the school year.

Mrs. Williams stayed in the hospital for 1 week after the completion of the ECT. At that time, her insurance ran out, and she was discharged home. Her discharged diagnosis was "major depression in remission."

Jeannette could see that her mom looked confused as she drove her home. She did not seem to recognize the neighborhood where she had lived for 30 years and raised her children. At home, her mother could not find the coffee or the sugar. She did not recognize the blender that Jeannette had bought her the previous Christmas.

A week later, Jeannette went to see the psychiatrist with her mother. The psychiatrist reassured her that he had never seen a case of permanent memory loss following electroshock, except for memory blanks for the period immediately around the shock treatment.

In September, 2 months after the ECT, Mrs. Williams tried to return to teaching but quit after 2 weeks. She could not remember the books or teaching materials she had been using for several years. The principal, who had started at the school a year earlier, looked like a stranger to her. She had trouble recognizing most of her previous students, including some who had been in music class with her for several years.

For the first time in her life, Mrs. Williams found she was having difficulty hearing music in her head. She was slow reading music and was

distraught that she could not learn new pieces by heart anymore. She felt like a beginner in music, except she could not learn as well as a beginner. She wanted to die and became suicidal for the first time in her life.

Jeannette took her mother back to the psychiatrist, who insisted that none of these problems could be from the shocks administered to her mother's head. He said that Mrs. Williams was depressed and needed more ECT. Instead, Jeannette took her mother home to live with her.

It was now January, and her mother was not getting any better. Mom was a changed person. Her personality was gone. So was her vitality. She could not remember the simplest things such as a phone call message or a list of three items to get at the grocery store.

Jeannette took her mom to the university medical center for evaluation. Lengthy neuropsychological testing over a 2-day period indicated that her mother had major impairments in anterograde memory (learning and recalling new material) and in retrograde memory (remembering past events). Some of her memory losses extended back several years. She had difficulty concentrating, and there were impairments of abstract reasoning. Formerly very quick mathematically, she was now poor at simple calculating. Her overall IQ had dropped 20 points. She became very fatigued and frustrated from the effort of trying so hard on the tests.

The neuropsychologist described the pattern as typical of traumatic brain injury, but after a consultation with Mrs. Williams's former psychiatrist, he avoided any suggestion that the deficits could have been caused by a series of electroshocks to the brain. Brain wave studies showed that Mrs. Williams had abnormal slow waves on her electroencephalogram (EEG) consistent with brain injury to the right frontal lobe and the anterior portion of the right temporal lobe (the two sites of electrode placement). A brain scan (MRI) showed possible atrophy in the same region.

To this day, Mrs. Williams's psychiatrist states that he has never seen a case of permanent memory loss, or any other permanent neuropsychological deficits, following ECT. He did not report the case in the literature, to the Food and Drug Administration (FDA), or to the manufacturer of the shock machine.

Mrs. Williams remains chronically depressed and refuses to go to any doctors for anything. She lives with her daughter, who supports her financially.

Cases like Mrs. Williams's have become increasingly common as psychiatry relies more and more exclusively on drugs and ECT. The last decade has seen a resurgence in the promotion and use of ECT, also called electroshock, or simply shock treatment. For a brief time before the 1997 edition of this book, the press had taken note of the escalating controversy surrounding its use (Boodman, 1996). A critical article by Cauchon (1995) in *USA Today* was followed up by a remarkable editorial

("Patients, Public Need," 1995), declaring that "the long-term effects can be devastating. They include confusion, memory loss, heart failure, and, in some patients, death." In more recent years, the shock doctors have been working hard to promote this barbaric treatment and have received less criticism from the media.

ECT is a treatment that originated in Italy in 1938 for producing convulsions in psychiatric patients. At the time, it was thought that convulsions induced by a variety of methods, including insulin coma and stimulant medication, were useful in treating psychiatric disorders, especially schizophrenia.

Nowadays, ECT is recommended for major depression, usually when other approaches have failed. However, some doctors quickly resort to it. Probably more than 100,000 patients a year in the United States are shocked. The majority are women, and many are elderly. Advocates of shock have resisted the creation or maintenance of state registers for shock treatment, so most of the data on the frequency of its use are relatively old. In California, for example, two-thirds of shock patients were reported to be women, more than half of whom were 65 or older (Department of Mental Health, 1989). Data (1989-1993) from Vermont concerning ECT showed that 77% of shock patients were female (W. Sullivan, personal communication, 1996). For all sexes, 58% were at least 65 years old, and 20% were at least 80 years old. During this time, one Vermont hospital, Hitchcock Psychiatric, shocked 35 women and 1 man who were 80 and older. Overall, the hospital shocked 112 women and 26 men during those 5 years.

The use of ECT tends to vary from institution to institution. At Johns Hopkins, for example, a biologically oriented center, 20% of the inpatients may be on a regimen of ECT at any one time (Wirth, 1991). The data was obtained under oath in a deposition, and I'm unaware of any more recent data, but shock treatment in general has increased in usage since then.

BREAKING NEWS IN ECT RESEARCH: SHOCK TREATMENT CAUSES IRREVERSIBLE BRAIN DAMAGE AND DYSFUNCTION

Beginning in 1979, when I published *Electroshock: Its Brain-Disabling Effects*, through the 1997 edition of *Brain-Disabling Treatments in Psychiatry*, and even until 2006, during my most recent trial testimony in an ECT malpractice case, I have had to marshal sophisticated, detailed, scientific arguments to show that shock treatment causes permanent memory loss and cognitive dysfunction. In presenting my evidence and my

conclusions, I had to overcome uniform disapproval and disagreement from the electroshock establishment that dominates the scientific discourse. Even psychiatrists who rejected ECT in their own practices would not risk standing up in opposition to the powerful ECT lobby.

Then something remarkable happened. In 2007, a team led by long-time, staunch electroshock advocate Harold Sackeim et al. published a follow-up study of patients given electroshock. The researchers found that the patients were devastated with widespread losses not only in memory, but also in cognitive functioning—the ability to think and learn.

Sackeim et al. (2007) followed up 347 patients given the range of currently available methods of electroshock, including the supposedly newer and most benign forms, and confirmed that electroshock causes *permanent* brain damage and dysfunction. The patients were selected from the community, that is, from patients in the real world of clinical practice rather than from an experimental study.

When tested 6 months after the last ECT, each form of treatment was found to cause lasting memory and cognitive dysfunction. The losses extended far beyond the erasure of memories surrounding a few months before and after the treatment. Many patients never recovered normal memory function. They described difficulties learning new things and suffered measurable losses on testing in “global cognitive status.” Although the authors avoided straightforward language, the patients were suffering from permanent brain damage affecting global mental function.

The results of the Sackeim et al. (2007) study were highly statistically significant ($p < .0001$ on 10 of 11 tests and $p < .003$ on the 11th). Adding to the evidence for permanent brain damage, many of the patients also had persistent EEG abnormalities 6 months after the treatments had ended. Although the older shock techniques were the most damaging, they were also the most commonly used in the community, and the newer technologies also produced significant lasting deficits in memory and cognitive function.

Despite Sackeim’s vigorous opposition to my views over the past many years, his study (Sackeim et al., 2007) cited my 1986 scientific article “Neuropathology and Cognitive Dysfunction From ECT” published in the *Psychopharmacology Bulletin*, noting that “critics contend that ECT invariably results in substantial and permanent memory loss.”

STILL AVOIDING THE FACTS

Remarkably, the detailed Sackeim et al. (2007) study leaves out some of the most important details, such as exactly what proportion of patients

suffered from each of the various deficits in memory and overall cognitive functioning. The tone of the article implies that just about everyone suffered from deficits; they are treated as one catastrophic group. But the all-important details were not disclosed. The extraordinarily low p -value on the cognitive testing ($p < .0001$) provided a strong indicator that the devastation was widespread, involving the vast majority of patients.

Sackeim et al. (2007) also failed to address the real-life impact of these losses on individual patients and did not provide any clinical vignettes. Stating that shock treatment permanently reduces memory and cognitive function, and describing it statistically, failed to capture the manner in which the "treatment" destroyed the minds of these patients and wrecked their lives. That is why I opened the chapter with the story of Sarah Williams.

Did his own research at last induce Harold Sackeim to make public statements withdrawing his previous wholehearted support for ECT? To the contrary, shortly after the publication of his paper I began to receive calls from the media asking me to respond to promotional claims by Dr. Sackeim in support of a supposedly new and improved form of ECT that sounded very much like the same old thing. One is left to wonder what drives so many mental health professionals in such an unrelenting, remorseless fashion to damage the brains of their patients.

MORE BREAKING NEWS IN ECT RESEARCH: SHOCK TREATMENT CAUSES SUICIDE

ECT is frequently justified as treatment of last resort in cases at high risk for suicide. But research uniformly shows that ECT has no beneficial effect on the suicide rate. Indeed, the most thorough study available, published in the *British Journal of Psychiatry* in 2007, found an overall *increased* rate of suicide in patients previously given ECT (Munk-Olsen et al., 2007). In addition, "patients treated with ECT in the past week had a *greatly* increased risk of suicide compared with other patients (RR = 4.82, 95% CI 2.22–10.95)" (p. 437, emphasis added).

The authors are proshock and minimized the importance of their results concerning increased suicide, not even mentioning it in the title. Furthermore, they failed to make clear that this data wholly contradicted the main justification for giving shock treatment: that it is supposedly the quickest and most effective way of preventing acute suicidal activity. Instead, without evidence the authors repeated the old saw that "suicidal intent in patients with depression is rapidly relieved by ECT" (p. 438).

Munk-Olsen et al. (2007) based their observation on ECT-induced suicidality on a review of all inpatient admissions to a Danish hospital

from 1976 to 2000 where 95% of the treatments were unilateral, indicating that the more modern techniques were used. Although the total number of patients given ECT was not provided, the numbers were considerable, given that 149 patients died by suicide during the study period.

All ECT studies involving larger numbers of patients are conducted by doctors who favor the treatment and therefore have access to the data, and invariably they minimize or misrepresent negative results. Munk-Olsen et al. (2007) are typical in this regard, not including any research critical of ECT in their bibliography. The study found that mortality from natural causes was also elevated during the first 7 days after ECT but that overall, it was decreased, especially for respiratory diseases. However, there is no discussion of death due to ECT treatment itself, including anesthesia, which in itself poses a significant risk (Lagasse, 2002).

In a blatantly misleading fashion, a series of negative studies were cited by the American Psychiatric Association (APA; 1990b) task force report as showing a positive effect. For example, a retrospective study by Avery and Winokur (1976) found no improvement in the suicide rate compared to matched controls who had no shock treatment: "In the present study, treatment was not shown to affect the suicide rate" (p. 1033). Yet it was presented in the 1990 task force report as supporting the position that ECT results in "a lower incidence of suicide" (p. 53). The task force also mentioned three other studies as supporting a beneficial effect on suicide. However, two of them (Avery et al., 1977; Milstein et al., 1986) specifically found no such beneficial effect, and the third (McCabe, 1977) did not even deal with suicide. Meanwhile, unmentioned were two retrospective studies of relatively large populations of ECT patients and matched controls in which ECT had no effect on the suicide rate (Babigian et al., 1984; Black et al., 1989).

I have rarely seen so much outright fabrication in the psychiatric literature as I have seen in regard to ECT and lobotomy (for more details, see Breggin, 1979, 1981a&b, 1982). Perhaps because these treatments are so violent and devastating, the doctors who perpetrate them, much like other perpetrators of violence (Breggin, 1992a), are especially prone to hide or to lie about the harmful effects of what they are doing.

Overall, there is little or nothing in the literature to suggest that ECT ameliorates suicide, whereas a significant body of literature confirms that it does not, and the most thorough study shows that it increases the overall suicide rate, including a major increase within the week after the last ECT. Once again, treatment opinions are not driven by empirical data. Instead, empirical data is ignored, distorted, or misrepresented to confirm treatment opinions.

My own clinical impression also confirms that ECT increases the suicide risk for many patients. After ECT many patients profoundly miss

memories of significant past events in their lives and feel overwhelmed by their inability to learn and to remember as well as they once did. Many feel as if their personalities and identities have been destroyed. As a result, they often feel deeply betrayed by their doctors. Inevitably some grow increasingly hopeless and suicidal. It is well known, for example, that Ernest Hemingway attributed his suicide to despair over ECT ruining his memory and rendering him unable to write (Hotchner, 1966).

As they attempt to recover from the treatment, ECT patients frequently find that their prior emotional problems have now been complicated by brain damage and dysfunction that will not go away. If their doctors tell them that ECT never causes any permanent difficulties, they become further confused and isolated, creating conditions for suicide.

Many shock survivors have told me that reading my articles and books about ECT was a life-affirming experience for them. Instead of reacting with more despair to the confirmation of their ECT-induced brain damage and disability, they have felt understood and empowered for the first time. Mental health professionals should be advised that it is both ethical and beneficial to acknowledge to patients in a supportive, empathic manner that they have been injured by the treatment.

ADDITIONAL BREAKING NEWS: ECT IS INEFFECTIVE

Ross (2006) recently reviewed the sham ECT literature: "The author reviewed the placebo-controlled literature on electroconvulsive therapy (ECT) for depression. No study demonstrated a significant difference between real and placebo (sham) ECT at 1 month posttreatment." This was the crowning summary of considerable prior research confirming that ECT is ineffective.

Rifkin (1988) noted that the claim is frequently made that ECT is more effective and works more rapidly than drugs in the treatment of depression. He found nine controlled studies comparing the two treatments, but they were badly flawed. He could find no conclusive evidence that ECT was better than antidepressant treatment.

Crow and Johnstone (1986), in a review of controlled studies of ECT efficacy, found that both ECT and sham ECT were associated with "substantial improvements" and that there was little or no difference between the two. Crow and Johnstone concluded, "Whether electrically induced convulsions exert therapeutic effects in certain types of depression that cannot be achieved by other means has yet to be clearly established" (p. 27).

Crow and Johnstone's (1986) critical review, which was presented at a large conference of shock advocates, is not cited in the APA report

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on ECT. Instead, the APA (1990b) task force's proposal for a "sample patient information sheet" declared that "ECT is an extremely effective form of treatment" (p. 160).

At the June 1985 Consensus Conference on ECT, critics and advocates of ECT debated the issue of efficacy. The advocates were unable to come forth with a single study showing that ECT had a positive effect beyond 4 weeks. Many studies showed no effect, and in the positive studies, the improvements were not dramatic. That the treatment had no positive effect after 4 weeks confirmed the brain-disabling principle since 4 weeks is the approximate time for recovery from the most mind-numbing effects of the ECT-induced acute organic brain syndrome or delirium.

The Consensus Conference panel concluded in its report that ECT had no documented positive effect beyond 4 weeks. Acute brain damage and dysfunction, with a high probability of permanent adverse effects, are inflicted upon the patient in order to achieve a brief period of traumatically induced emotional blunting or euphoria. ECT is a wholly irrational, unjustifiable treatment.

ANOTHER DRAMATIC EVENT IN THE WORLD OF SHOCK TREATMENT

For several decades, I have been a medical expert in lawsuits against doctors and hospitals for causing permanent brain damage with electroshock treatment. I have also been an expert in product liability suits against the manufacturers of the machines. A number of the suits against doctors, hospitals, and shock manufacturing companies were resolved, often with substantial settlements for the victims. But on several occasions, when cases against doctors went to trial, they were lost. The cases in which I testified were not the only ones that failed to win a jury verdict. Until 2006, not a single electroshock malpractice case had ever been won in court anywhere in the world.

Why were the cases lost in trial? There are no easy answers. In several of the cases in which I was involved, our side presented two, three, and even four medical experts who confirmed that shock causes brain damage. At the same time, the defendants could always find well-known professors of psychiatry to defend the treatment as essentially harmless and enormously beneficial. Probably it has been hard for juries to disentangle totally conflicting evidence from critics and advocates of the treatment. In addition, critics like me refuse to send patients for shock treatment, and of course, we do not administer it to patients, so the advocates can present themselves as the only experts with the "clinical experience." In addition, it must be hard for juries to believe that so many doctors and so

many medical groups would support a treatment that routinely damages the brain. They must find it hard to believe that doctors would simply lie about the damaging effects of their treatments. Finally, victims of shock treatment often remain irritable and angry for the rest of their lives, suffering from the emotional instability and poor impulse control associated with brain damage and dysfunction. As a result, they sometimes present unsympathetically when they testify before juries.

Finally, in 2006, an electroshock case was won against a physician. But even then, the verdict was quirky. The jury found the prescribing physician negligent. He was the one who initially recommended the treatment. But it exonerated the physicians who administered the treatment, even though they broke numerous standards, including giving the treatment on an outpatient basis on a much more frequent basis than is usually done in the hospital. I thought the doctors who carried out the treatment in such an excessive and cavalier manner were far more to blame than the doctor who recommended it.

The case involved a nurse who believed she had previously benefited from the treatment. This time, the series of closely packed treatments obliterated her nursing training and her personal memories extending back years and caused continued memory and cognitive dysfunction. I cannot explain why this case was won, while so many others have been lost. In most of the prior ECT trials, I was one among several experts testifying on behalf of the victim; but this time I was by myself. However, the patient's psychotherapist, an empathic and courageous woman, described the devastating effects of the treatment on her client. The attorney was excellent; but I have worked with good attorneys on earlier shock suits. A key defense expert in many cases, Max Fink (see subsequent discussion), was not called to the witness stand, and this probably hampered the doctors' case. Fink had admitted in deposition that he had not read the victim's medical record but that he had already decided to testify on behalf of the doctors that they had done nothing wrong. It seemed to compromise his credibility and perhaps kept the defense from calling him to the stand. Whatever the reasons for this victory, in the future, medical experts who are critical of shock treatment will now be armed with Sackeim et al.'s (2007) research, creating a major breach in the professional wall of silence about shock's damaging effects.

THE FOOD AND DRUG ADMINISTRATION AND ECT

In 1979, the FDA classified shock devices as demonstrating "an unreasonable risk of illness or injury" (see Food and Drug Administration [FDA], 1990). This would have required animal testing for safety. However, under

pressure from the APA, the FDA gave notice of its intent to reconsider its original decision and to reclassify ECT machines as safe. The APA's (1990b) task force report was timed to come out in the midst of the FDA's political squirming over ECT.

The FDA's (1990) final report reads remarkably like the APA's (1990b) report, including the mistaken or false citations mentioned earlier in this chapter. Although no large animal studies have been done with shock devices since the 1950s (some have been done with rats) and although those earlier large animal studies consistently demonstrated brain damage (see subsequent discussion), the FDA panel recommended defining ECT devices as safe for depressed patients. It did so ambivalently, recommending that the approval be delayed until the establishment of engineering safety standards for the machines. The approval process continues to be delayed by the lack of approved standards, and ECT exists in a kind of FDA limbo, which has not discouraged psychiatrists from using it.

I have reviewed what the FDA has made available through the Freedom of Information Act as its complete file on ECT. There are dozens of recommendations from state-funded and private patient rights and advocacy groups to ban ECT, and hundreds more from patients who feel that they have been permanently damaged by the treatment. It is astonishing that the FDA has ignored or rejected such an avalanche of official recommendations and personal reports and protests.

In recommending the approval of ECT as safe and effective, the FDA ignored a most remarkable situation. Before being put on the market, the ECT machines, such as the commonly used MECTA, were not tested for safety on animals or humans. There were no systematic or controlled studies to evaluate their impact on the living brains of animals or humans. The FDA simply took the word of organized psychiatry and ECT advocates that the treatment is safe and effective. Once again I am left to wonder if we are dealing with a treatment that is so egregiously abusive that the perpetrators, including the APA and the FDA, feel compelled to hide the facts from the public.

THE POLITICS OF THE 1990 AMERICAN PSYCHIATRIC ASSOCIATION REPORT

The political nature of the APA (1990b) task force report is reflected in the membership of the panel that wrote it. The chairperson, Richard Weiner, was APA's official representative in defense of ECT at the FDA hearings and has for some time been APA's chief spokesperson on the subject. Two of the other six members are psychiatrist Max Fink and psychologist Harold Sackeim, whom we have already met as among the

nation's most zealous promoters of the treatment. Fink (1994, 1995) has actively pressed for the increased use of shock treatment for children and adolescents. Sackeim et al. (1993) wrote an article calling for a return to much higher electrical doses, given the "old-fashioned way," with bilateral electrode placement (see subsequent discussion) to increase the intensity of the shocks.

By contrast, the task force (APA, 1990b) sought no input from the several patient organizations that oppose the treatment, and none from psychologists, psychiatrists, neurologists, and other professionals who are critical of it.

The APA (1990b) task force report, in its acknowledgments, thanked the manufacturers of electroshock machines for their contributions; company advertising handouts are listed as useful sources of public information; and the names, addresses, and phone numbers of these companies are provided in the report. The task force is particularly positive toward Somatics Inc., whose sole function is to manufacture the electroshock machine Thymatron. Somatics Inc. is acknowledged for providing "input into the guidelines." Under the heading "Materials for Patients and Their Families," the task force cited a pamphlet by Richard Abrams and Conrad Swartz and a videotape by Max Fink, both of which are advertising materials for Thymatron and can only be obtained by writing to the manufacturer.

The report (APA, 1990b) nowhere mentions any link between Thymatron and Richard Abrams, who would appear to be the task force's most valued expert. One of Abrams's articles is recommended under "Materials for Patients and Their Families" and another under "Materials for Professionals." Nine of his publications are cited in the report's general bibliography, making him by far the most heavily represented author. Abrams is also listed among those individuals who "provided comment on the draft of the ECT Task Force Report." However, his most interesting affiliation is unmentioned: Abrams owns Somatics Inc. In a deposition in which he was a medical expert (*DeToma v. Brohamer*, 1991), as a result of my prompting the defense attorney to ask the question, Abrams had to acknowledge under questioning that Somatics Inc. is the source of 50% of his income.

ECT, WOMEN, AND MEMORY LOSS

Women have always been the main victims of the most destructive psychiatric treatments, including lobotomy. In recent decades, older women have become the major population for ECT, despite the absence of controlled studies on safety or efficacy in the elderly.

One of the most remarkable reports in the ECT literature was published by Warren (1988), who studied 10 women post-ECT, including their family relationships. Many of the women thought that the purpose of the treatment was to erase their memory. While some felt it was helpful to forget painful memories, they “uniformly disliked the loss of everyday memory, as well as associated effects such as losing one’s train of thought, incoherent speech, or slowness of affect. What specifically was forgotten varied from matters of everyday routine to the existence of one or more of one’s children.” Warren is not a physician and perhaps without knowing about the specific clinical syndrome, she described mild to moderate dementia caused by closed-head injury in the form of ECT.

According to Warren, family members sometimes approved of the memory loss. One husband said, “They did a good job there,” referring to his wife’s loss of memory concerning their past marital conflicts. A patient who had been molested by her mother’s brother believed that her mother wanted her to have “the full treatment” to “make me forget all those things that happened.”

Three of the 10 women lived in dread of ECT for years afterward but were afraid to express their angry feelings for fear of being sent back to the hospital for involuntary shock treatment. In my clinical experience, this is a realistic fear. Doctors frequently respond to complaints about the treatment by deciding that the patient is in need of more treatment. Repeated “treatment” can usually be relied on to put an end to all protests.

Shock treatment has been used even more blatantly to erase the memories and even the personalities of patients, usually women. H. C. Tien, in the early 1970s, described the use of unmodified ECT to erase the personalities of women, then to “reprogram” them as more suitable wives—with their husbands’ help (“Electroshock,” 1972; “From Couch to Coffee Shop,” 1972). World-renowned Canadian psychiatrist D. Ewen Cameron at McGill University, in part utilizing secret funds from the Central Intelligence Agency, used multiple ECTs to obliterate the minds of his patients and then to reprogram them (Cameron et al., 1962; for more details on the Tien and Cameron controversies, see also Breggin, 1979, 1991b).

ECT AND THE ELDERLY

As already noted, elderly women have become the most frequent target of ECT. The elderly, of course, have more fragile brains and are especially sensitive to biopsychiatric interventions, even relatively mild doses of drugs. In addition, many elderly already suffer from memory dysfunction

due to a variety of causes, making them especially vulnerable to the worst effects of ECT.

Against all common sense, the APA (1990b) task force advised that ECT can be used "regardless of age" (p. 15) and cited the successful treatment of a patient aged 102 (pp. 71-72). It did warn, however, that "some elderly patients may have an increased likelihood of appreciable memory deficits and confusion during the course of treatment" (p. 72).

The aged are, in fact, gravely at risk when exposed to any form of head trauma, including electrically induced, closed-head injury from ECT. There are a growing number of reports of special dangers to the elderly that were not mentioned in the APA (1990b) or FDA (1990) reviews (Figiel et al., 1990; Pettinati et al., 1984). In a curious twist, an article by Burke et al. (1987) was listed in the bibliography of the APA report but not cited in the actual discussions of the elderly. Burke et al. found a high rate (35%) of complications among the elderly. They noted, "Common complications in the elderly include severe confusion, falls, and cardiorespiratory problems" (p. 516).

In a study involving 3 times as many women as men, Kroessler and Fogel (1993) produced data indicating that ECT can cause a devastating decline in longevity:

This is a longitudinal study of 65 patients who were 80 years old or older at the time they were hospitalized for depression. Thirty-seven were treated with ECT and 28 with medication. Survival after 1, 2, and 3 years in the ECT group was 73.0%, 54.1%, and 51.4% respectively. Survival after 1, 2, and 3 years in the non-ECT group was 96.4%, 90.5%, and 75.0% respectively. (p. 30)

These are extraordinary findings, indicating a very high increase in mortality in the elderly who received ECT. The authors, however, argued that the patients receiving ECT were more physically ill and hence at greater risk of dying. They provided no data to justify this speculation or to otherwise explain such a vast difference in mortality.

In the Kroessler and Fogel (1993) study, the tragic lethality of ECT was compounded by its lack of efficacy. ECT patients were much more frequently rehospitalized for depression than non-ECT patients (41% vs. 15%). The recurrence rate of depression was more than twice as high among the ECT patients compared to the non-ECT patients (54.1% vs. 25%). Lasting recovery from depression was much lower in ECT patients (22% vs. 71%). If psychiatry were practiced in a rational manner, a study like this would have brought a halt to giving ECT to the elderly.

Elderly women are particularly vulnerable to being diagnosed with depression, with the associated risk of having ECT imposed upon them.

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Older women often have many reasons—psychosocial and economic, some of them rooted in the ageist and sexist attitudes of our society—for feeling depressed. Often, these women need improved medical care, social services, family involvement, and loving care from friends and volunteers. Too often, their depression is being caused or aggravated by multiple medications for elevated hypertension or elevated cholesterol that can cause feelings of fatigue and depression. Even the so-called antidepressants that have been given to them prior to ECT can cause suicidal depression and an overall worsening of their mental state. Instead of ECT, they need their medications and their overall health care reevaluated, along with all of their basic needs. Meanwhile, they typically do not have the strength to resist a doctor's proposal that they undergo electroshock. There may be no family members available or willing to protect them. One thing the elderly do not need is more brain cell death, mental dysfunction, and memory deficits.

I have been a consultant or a medical expert in several suits in which psychiatrists have tried to administer electroshock against the will of elderly women who had no family to defend them. Each time, the doctors have backed down or, as in the case of Lucille Austwick, they have lost in court (Boodman, 1996). However, many other elderly women are probably getting shocked involuntarily without their situation gaining public attention. In addition, in my experience, many seemingly voluntary patients are badgered or misled into taking the treatment.

BRAIN INJURY BY ELECTROSHOCK

The Production of Delirium (Acute Organic Brain Syndrome)

After one or more shock treatments, ECT routinely produces delirium or an acute organic brain syndrome. Abrams (1988), although an advocate of the treatment, has himself observed that

a patient recovering consciousness from ECT understandably exhibits multiform abnormalities of all aspects of thinking, feeling, and behaving, including disturbed memory, impaired comprehension, automatic movements, a dazed facial expression, and motor restlessness. (pp. 130–131)

At times, patients are so organically impaired following ECT that they will sit around apathetically on the ward, unable to engage in any activities. On occasion, the patients' neurological dilapidation from routine ECT will reduce them to lying in a fetal position for many hours. In malpractice suits in which I have been a medical expert for plaintiffs,

psychiatrists for the defense have claimed that this kind of neurological collapse following ECT is normal and harmless.

Given that ECT routinely produces acute, marked brain dysfunction, there can be no real disagreement about its damaging effects. The only legitimate question is, "How complete is recovery?" Even without all the confirmatory evidence presented in this chapter, basic neurology warns that it will frequently be incomplete.

ECT As Closed-Head Electrical Injury

Neurology recognizes that relatively minor head trauma—even without the delirium, loss of consciousness, and seizures associated with ECT—frequently produces chronic mental dysfunction and personality deterioration (Bernat et al., 1987). If a woman came to an emergency room in a confusional state from an accidental electrical shock to the head, perhaps from a short circuit in her kitchen, she would be treated as an acute medical emergency. If the electrical trauma had caused a convulsion, she might be placed on anticonvulsants to prevent a recurrence of seizures. If she developed a headache, stiff neck, and nausea—a triad of symptoms typical of post-ECT patients—she would probably be admitted for observation to the intensive care unit. Yet ECT delivers the same electrical closed-head injury, repeated several times a week, as an alleged means of improving mental function. ECT is electrically induced closed-head injury.

The symptoms of mild to severe closed-head injury were listed in detail by Fisher (1985). They include impairment of every area of mental, emotional, and behavioral function, and confirm that the multiple adverse effects of ECT on the mind and brain are classic symptoms of closed head injury. McClelland et al. (1994) described the postconcussive syndrome in terms of

the emergence and variable persistence of a cluster of symptoms following mild head injury. Common to most descriptions are somatic symptoms (headache, dizziness, fatiguability) accompanied by psychological symptoms (memory and concentration difficulties, irritability, emotional lability, depression and anxiety).

The authors observed that between one-third and one-half of head injury victims experience this symptom cluster over the first few weeks and a "substantial minority" continue to experience it for months or a year or more.

Head injury victims, including post-ECT patients, frequently develop an organic personality syndrome with shallow affect, poor judgment,

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irritability, and impulsivity. They seem "changed" or "different" to people around them, much as lobotomy patients often seem to their families. Sometimes they become slightly clumsy, moving awkwardly or dropping things. Often they have "lapses" where they cannot think or cannot voice their thoughts. Sometimes their handwriting deteriorates. Headaches frequently begin with the traumatic treatment and may recur indefinitely.

Many post-ECT patients suffer from irreversible generalized mental dysfunction with apathy, deterioration of social skills, trouble focusing attention, and difficulties in remembering new things. I have worked with a number of them who suffer from dementia, confirmed by neuropsychological testing. Several have developed partial complex seizures or psychomotor epilepsy, permanently abnormal EEGs, and atrophy on brain scans. Many have been deprived of the experience of years of their lives, their professional careers, and their mental ability following ECT (Breggin, 1979, 1981a).

Death, Suicide, and Autopsy Findings

Many deaths were reported in association with ECT in the first few decades of use. An extensive autopsy series indicated that many suffered from trauma to the brain resulting in visible pathology (Impastato, 1957). Advocates for ECT have claimed the death rate is very small or nearly nonexistent; but I have suspected that deaths are simply no longer reported. For example, I know of deaths of ECT recipients in the Baltimore-Washington, DC, area that have gone unreported.

There has been some epidemiological confirmation of the probability of a significant death rate. A law passed in Texas in the early 1990s required the reporting of death within 2 weeks after ECT. From June 1993 through August 1994, 8 deaths were reported among nearly 1,700 patients subjected to shock treatment. Controversy surrounds causation, and critics of ECT attempted without success to obtain more autopsy details (Smith, 1995).

Memory Deficits

Electroshock specialists almost never seriously consider the memory deficits of their patients. In case after case that I have evaluated for clinical or forensic purposes, I have been the first doctor to take the symptoms seriously, let alone to take a complete inventory of memory losses and ongoing mental difficulties. I have previously outlined a method for evaluating memory deficits from ECT (Breggin, 1979).

The recent study by Sackeim et al. (2007) described earlier in the chapter should put to rest the question of whether or not ECT causes

permanent cognitive dysfunction and memory loss. However, psychiatry has a long history of ignoring negative research about its treatments.

For example, the APA (1990b) task force report, like the FDA (1990) report, disregarded all of the relevant research on memory loss, except for Freeman and Kendell's (1986) study, which the task force mentions and then grossly misrepresents. That study asked patients to assess their memory function a year or more after electroshock treatment. The authors themselves remarked that the study was biased toward a low reporting of memory dysfunction because the patients were interviewed by the same doctor who had treated them. Nonetheless, 74% mentioned "memory impairment" as a continuing problem, and "a striking 30% felt that their memory had been permanently affected." In defiance of the facts, the APA (1990b) task force cited Freeman and Kendell (1986) as indicating that "a small minority of patients, however, report persistent deficits."

Squire and Slater's (1983) study, also omitted by the APA (1990b) task force, found that 7 months after treatment, patients reported an average loss of memory spanning 27 months. Squire, in a personal communication to me at the June 1985 Consensus Conference on ECT, explained that one patient lost the recollection of 10 years of her life. He told me that he felt it was not necessary to report this in his actual publication.

The Consensus Conference on ECT (1985) used Squire and Slater's (1983) results to conclude that "on average, patients endure memory loss extending from 6 months prior to the treatment to 3 months afterward." These data, while serious enough in themselves, are misleading. The data reported at 7 months following treatment, cited in the above paragraph, are more likely to be accurate. The brain cannot regenerate lost brain cells or lost memories. With the passage of more time, there is little likelihood of increased improvement, but much likelihood of a growing tendency to deny the losses.

The APA (1990b) task force also ignored older controlled clinical studies by Janis (1948, 1950; Janis et al., 1951) showing extensive, permanent loss of important personal memories and life history following routine ECT. Janis (1948, 1950; Janis & Astrachan, 1951) interviewed 19 patients before and after routine ECT, and 11 control patients with similar diagnoses in the same hospitals. The results 1 month postshock were striking: Every shock patient had significant memory losses. Many patients were unable to recall 10–20 life experiences which had been available to recall prior to electroshock treatment.

Janis (1950) followed up five of the patients at 2.5–3.5 months later. Most of the lost memories remained lost. Another follow-up 1 year later showed continuing losses (see review in Breggin, 1979).

The data generated by Janis (1948) confirmed the importance of ECT spellbinding with denial and anosognosia. Patients tended to minimize or even confabulate to cover up their memory losses, rather than to exaggerate them. One patient, for example, in his pre-ECT interview, reported that he had been unable to work for several months prior to coming to the hospital. The historical facts were confirmed by the family. But after 12 ECTs, he was unable to recall the period of unemployment. Instead, he claimed that he worked right up to his hospitalization. As Janis confirmed, patients often do not complain spontaneously to doctors about their memory loss; they tend to deny it.

Not only was Janis's research left out of the 1990 APA report, but over the years, his work has been wholly misrepresented by shock advocates. Two of the more important reviews commonly read during my psychiatric training actually cited Janis as evidence that ECT did not harm memory (reviewed in Breggin, 1979).

In 1986, Weiner et al. attempted to measure the loss of personal subjective recollections following ECT because these are "most consistent with the nature of memory complaints by ECT patients themselves." The memory inventory in the study spanned several years prior to the shock treatment. The group found "objective personal memory losses" that lasted through the 6-month duration of the study.

In an earlier article by a team that also included Weiner (Daniel et al., 1982), there was emphasis on the potentially injurious effect on the patient and the patient's family of losing autobiographical memories. The authors observed that "autobiographical memory failures, if added across a course of ECT, may produce gross autobiographical memory gaps that may be disconcerting to a patient and a patient's family, because the patient's sense of continuity with his or her own past may be disrupted" (p. 923). Yet their subsequent study, in which they demonstrated the existence of the autobiographical memory losses, failed to mention how distressing they can be (Weiner et al., 1986).

One of the newer techniques of shock treatment—multiple monitored electroconvulsive therapy (MMECT)—employs four electroshocks in one session, while recording EEG, electrocardiogram, and vital signs. Barry Maletzky, an advocate of the treatment, is one of the few who have asked patients in detail about their memory function following ECT. After pointing out that psychological testing has sometimes failed to confirm cognitive deterioration (Maletzky, 1981), he observed,

However, if one listens to what patients say who are treated with either conventional ECT or MMECT, subtle cognitive deficits, not easily tested, are discussed. Some patients will mention deficits only if careful inquiry is pursued. Most will not identify these problems even if asked,

thus indicating that either they are absent or so subtle as to be imperceivable to the patient. (p. 180)

Maletzky (1981) then goes on to describe a series of 47 MMECT patients who were interviewed 3–6 months after ECT treatment. Thirty-six percent identified a cognitive problem, including difficulty finding their way around, recalling past events in sequence, and understanding TV shows. In another ECT follow-up study by Maletzky (1981) reported in the same book, patients were given a questionnaire and interviews and 23% reported “long-term memory deficits.” The problems described by Maletzky’s patients extend beyond memory dysfunction to substantial cognitive deficits such as a math student’s loss of his ability to do computations in his head.

Devanand et al. (1994), in their review, skated over the surface of the many cognitive studies, dismissing most of them, failing to mention any of the Janis studies, ignoring follow-up studies indicating that patients frequently experience permanent memory loss, and raising no issues about the improbability of full recovery from traumatic acute organic brain syndromes. Appearing in the *American Journal of Psychiatry* amid growing controversy surrounding ECT, Devanand et al.’s (1994) review was seemingly intended as an establishment response to criticism. For this reason, I shall examine its conclusions at relevant points in this chapter.

STUDIES OF BRAIN DAMAGE FROM ECT

The recent study by Sackeim et al. (2007) that found widespread, persisting generalized cognitive dysfunction provides proof that ECT causes brain damage. There is also an extensive literature confirming brain damage from ECT. The damage is demonstrated in many large animal studies, human autopsy studies, brain wave studies, and an occasional CT scan study.

Animal and human autopsy studies show that shock routinely causes widespread pinpoint hemorrhages and scattered cell death. While the damage can be found throughout the brain, it is often worst beneath the electrodes. Since at least one electrode always lies over the frontal lobe, it is no exaggeration to call electroshock an electrical lobotomy.

In 1976, Friedberg published the first review of brain damage from ECT. This was followed by my own detailed critiques (Breggin, 1979, 1981a, 1986). None of these studies and none of the reviews on brain damage were mentioned in the 1990 APA task force report.

The original animal studies are from the 1940s and 1950s, but they are still valid. Several of them were elegant by any scientific standard.

The model for these studies was conducted by Hans Hartelius on cats and published in 1952 in a book-length publication titled "Cerebral Changes Following Electrically Induced Convulsions."

In the double-blind microscopic pathology examination, Hartelius (1952) was able to discriminate between the eight shocked animals and the eight nonshocked animals with remarkable accuracy. The experimental animals showed vessel wall changes, gliosis, and nerve cell changes:

The *vessel wall changes* found more frequently and more distinctly in the animals subjected to ECT consist of characteristic sac-like dilations of the perivascular spaces, which in some cases contain histiocytic elements. The *glial reaction*, of the progressive type, consists of an increase in the number of the small glial elements in the parenchyma and satellitosis beside the nerve cells. The *nerve cell changes* observed are in the form of various stages of chromophobia, frequently with coincident nuclear hyperchromatism. The arrangement of such cells is mainly focal.

The changes were statistically significant. Confirming their basis in sound pathology, the abnormalities were found most heavily in the animals given the greater numbers of ECTs, were most dense in the frontal lobe, and were correlated with increased age of the animal (implying increased vulnerability).

Hartelius (1952) was cautious in his determination of irreversibility. He required the detection of shadow cells and neuronophagia (the removal of dead or diseased nerve cells by phagocytes). On the basis of these findings, he concluded, "The question whether or not irreversible damage to the nerve cells may occur in association with ECT must therefore be answered in the affirmative."

Hartelius (1952) used relatively small doses of ECT. In fact, the amount of electrical energy he used was a fraction of that currently applied to the heads of shock patients. In general, however, animals are less susceptible to electroshock trauma to the head than humans and require more intensive electrical currents to achieve the same degree of damage. If given the doses used in clinical practice, the damage to the cats would almost certainly have been even greater.

Ferraro et al. (1946, 1949), of Columbia University and the New York State Psychiatric Institute, conducted controlled studies involving clinical doses of ECT on rhesus monkeys. The researchers used regular ECT machines, smaller-sized electrodes to fit the monkey heads, restraint to keep the heads from banging, and the minimally necessary dose of electricity to cause a convulsion, thereby approximating the intensity of current and voltage used to treat human beings (Ferraro and Roizen, 1949). The total energy dose was less than that routinely used in modern ECT.

In the 1946 study, Ferraro and Helfand administered ECT three times per week to the monkeys in relatively short courses of 4 to 18 in number. As a result of only 4 ECT, one animal had microscopic findings: "Here and there in the cerebral cortex there were some areas of rarefaction [cell loss]." After 12 ECT, another showed "small areas of rarefaction" as well as other evidence of cell deterioration and death. Another, again after 12 ECT, displayed "slight rarefaction of nerve cells and a few acellular areas in the front lobes." In addition to areas of cell death, they also found cells in various states of degeneration, loss of myelin sheaths, glial proliferation, dilated blood vessels, microscopic effusions of blood, petechial hemorrhages, and other neuropathology that they associated with the ECT. The pathological findings were roughly proportional to the numbers of ECTs. Their overall findings were very consistent with, although more severe than, those reported by Hartelius in cats.

In their 1949 study, Ferraro and Roizin. used larger numbers of ECTs (32-100). Although excessive by some standards in psychiatry, many patients in fact receive such larger numbers of shock treatments, usually spread over a number of years. After the fewest electroshocks, the researchers found evidence of cell death in the form of "moderate nerve cell rarefaction" and "acellular areas, again proportionate to the current intensity and the number of ECT." Photographs of the microscopic findings were reproduced in both papers.

Alpers and Hughes (1942a) studied the effects of ECT on cats and found evidence of subarachnoid hemorrhages and scattered punctate hemorrhages in the brain. They correlated this damage with autopsy findings in two human cases (Alpers and Hughes, 1942b). Alpers (1946) reviewed the literature on ECT experiments involving animals, including additional studies of cell death in dogs (Neuberger et al., 1942) and rabbits (Heilbrunn et al., 1942). Alpers noted that even studies that claimed to show little or no effects from ECT in fact often provided evidence of cellular abnormalities and even cell death in the brain.

Neither the Hartelius (1952) study nor any of the other studies using large animals cited in this section were included in the 1990 APA task force report on ECT. An oversight such as that cannot occur by chance but instead must have reflected a conscious attempt to withhold vital information about the dangerousness of ECT.

The Russians carried out a variety of neuropathology studies on animals subjected to clinical ECT to determine if there is permanent brain damage. Babayan called for a ban on the treatment in 1985, citing work at the USSR Academy of Medical Sciences as "convincing proof... pointing to grave changes in the central nervous system, the nerve cells, the glial-tissue apparatus" (p. 37). At another institute, studies of the brains of animals led to a "drastic reduction in the use of electroshock

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therapy in clinical practice" (p. 134). Babayan compared the treatment to lobotomy.

There have been no studies of large animals using modified ECT under clinical conditions, even though this so-called new form of ECT was developed in the 1960s. Meldrum and Brierley (1973) studied *drug-induced* (bicuculline) lengthy seizures in baboons and found widespread ischemic (due to lack of blood flow) changes. Meldrum et al. (1973) repeated their earlier experiment, now employing modified ECT, and found similar but lesser ischemic changes in neurons. They concluded that modifying the ECT gave some incomplete protection. However, the seizures were very long. Meldrum et al. (1974) once again studied the impact of drug-induced (allylglycine) seizures in baboons under modified conditions. They used 13 animals, and in 8, the seizures were brief, recurring 6–63 times in 2–11 hours, followed by recovery. The short-duration seizures produced no detectable pathology.

Templer (1992) reviewed the question of ECT and permanent brain damage. In regard to animal studies, he focused on Hartelius (1952) and also pointed out that animals given artificial ventilation (modified ECT) in other studies also had "brain damage of somewhat lesser magnitude."

While few psychiatrists are willing to admit in public that ECT causes brain damage, a large survey of the APA membership, conducted with anonymity in the 1970s, showed that 41% of the respondents agreed with the statement "It is likely that ECT produces slight or subtle brain damage." Only 26% responded that it did not (APA, 1978).

As noted previously, Devanand et al. (1994) published an article titled "Does ECT Alter Brain Structure?"¹ They concluded that animal studies do not show brain damage. They did this by dismissing the best studies. Hartelius (1952), for example, was criticized for applying a series of four ECTs, with each one spaced at 2 hours. But there is no reason to assume that this method is more damaging than larger numbers of shocks spaced over longer intervals. As currently used, multiple-monitored ECT inflicts four shocks within the space of an hour or so. In addition, it is extremely misleading of Devanand et al. (1994) to focus on that one group of animals. Some of Hartelius's animals, for example, were given one ECT per day for 4 days, others were treated "with clinical frequency" (three per week), and many showed evidence of brain damage.

Devanand et al. (1994) dismissed Ferraro and Roizen (1949) for using a "large number of ECSs [electroconvulsive shocks] relative to clinical practice," but in fact, many patients are given 32 or more treatments, sometimes in one series, more often in several. Ferraro et al. (1946), utilizing fewer shocks, were dismissed on the speculation that the current went through the brain stem.

Devanand et al. (1994) did not deal with the fact that almost every study using large animals, by their own table, showed damage. My review indicated that even purportedly negative studies, on actual reading, indicated harmful effects (Breggin, 1979). For example, Devanand et al. (1994) described Lidbeck's (1944) three dogs as developing "minimal perivascular and ischemic changes." They left out that in two of the four animals, "nerve cells were shrunken and there was a decrease in the number of stainable granules" (Lidbeck, 1944). Nor did they mention that one of the animals developed blood clots in its brain.

Even if Devanand et al. (1994) had valid points to make, criticizing a raft of animal studies that show damage cannot be used as a method for proving the safety of ECT. To be ethical and scientific, shock advocates would have to produce carefully conducted, large-animal studies that show no damage. In fact, the only studies that Devanand et al. (1994) found acceptable were performed on rats, rather than dogs, cats, and primates, whose brains are more akin to humans and more sensitive to damage. In comparison to monkeys, cats, and dogs, rats, with their smaller brains and thick skulls, are notoriously resistant to head trauma.

The prospects of more modern ECT being safe are nil. The newer methods add the risk of anesthesia, often complicated by multiple psychiatric drugs administered simultaneously. The electrical trauma must be sufficient to cause a grand mal seizure. Grand mal seizures, when repeated and especially when as severe as those caused by ECT, are in themselves harmful to the brain. Nor are modern variations in current intensity necessarily more benign because, in order to cause a seizure with the weaker currents, exposure time is often increased by 10-fold or more over earlier ECT methods. Also, in order to overcome the anticonvulsive effects of the sedatives administered to put the patients to sleep, modern ECT often inflicts more intense electrical energy on the brain than the older animal studies and older forms of ECT (see the section "Modified ECT"). Perhaps most obvious and important, the study by Sackeim et al. (2007) shows that the effects of modern ECT continue to be devastating.

In addition to demonstrating safety, shock advocates would also have to prove efficacy through double-blind clinical trials comparing ECT to sham or placebo in which the subject is put to sleep without the actually administering the shock. Thus far, placebo-controlled trials have failed to show any significant superiority of ECT over sham ECT.

Brain Scans

There has been contradictory evidence of ECT damage in brain scan studies, most of which have been carried out by staunch advocates of the

treatment. Using CT scans, Weinberger et al. (1979) found that chronic patients with schizophrenia who had ECT had more enlargement of their ventricles (cerebral atrophy) than those who had no ECT. Stretching to exonerate ECT, they declared, "Either ECT further enlarged the ventricles of the patients treated with it, or it was used with greater frequency in patients who tended to have larger ventricles." In another CT study, Calloway et al. (1981) found a correlation between frontal lobe atrophy and ECT in 41 "elderly depressives."

Coffey et al. (1991), using MRI, studied 35 patients before and after ECT. The follow-ups were 2 or 3 days after and 6 months after. In five subjects, they found "an apparent increase in subcortical hyperintensity." Coffey, a strong ECT advocate who has performed shock on many patients, dismissed his own finding as "most likely secondary to progression of ongoing cerebrovascular disease during follow up." I have seen several other patients with very similar post-ECT MRI findings.

Pande et al. (1990) found no MRI pathology in seven ECT patients. However, the studies were performed 1 week after the last ECT so that late-maturing pathology would not have been discovered. Bergsholm et al. (1989) found no pathology on MRI in 40 patients, with the exception of a 69-year-old man, who suffered a dilatation of the left temporal horn, which the authors dismissed as unrelated to ECT.

Devanand et al. (1994) reviewed the brain scan literature and found the evidence for brain damage unconvincing. They accepted Coffey et al.'s (1991) unsubstantiated claim that the four damaged patients had progressive cerebral vascular disease, rather than ECT pathology. They dismissed studies showing damage.

In reality, brain scans are not an appropriate instrument for measuring ECT brain damage. None of the damage found in the large-animal studies—such as small areas of dead and dying cells and small pinpoint hemorrhages scattered throughout the brain—would show up on brain scans, which cannot detect damage at a microscopic level until it is massive enough to result in gross atrophy or tissue shrinkage. To use brain scans to show that ECT is harmless is a scientific scam. On the other hand, in my medical-legal work I have on occasion seen patients whose before-and-after brains scans did detect atrophy following ECT.

MODIFIED ECT

For the past 40 and more years, a modified form of ECT has been standard, involving sedation with a short-acting barbiturate, muscle paralysis with a curare derivative or similar drugs that prevent activation of the muscles of the body, and artificial respiration with oxygen. The purpose

of these modifications was not, as some advocates claim, to reduce memory loss and brain damage. Muscle paralysis was intended to prevent fractures from severe muscle spasms, while the artificial respiration kept the paralyzed patient breathing.

The modifications used in contemporary ECT make it clear that ECT-induced convulsions are far more severe than the spontaneous convulsions in grand mal epilepsy. Patients with spontaneous seizures of unknown origin, or with seizures due to brain injury, rarely break their limbs or their vertebrae during the convulsion. The muscle spasms are not intense enough to produce these dramatic effects. Yet these fractures were common with unmodified ECT.

Shock advocates claim that newer modifications have made the treatment much safer and that its negative public image is unfairly based on the older methods. However, the most basic modifications—anesthesia, paralysis, and artificial respiration—are not new at all. I prescribed and administered this kind of modified treatment more than four decades ago (1963–1964) as a resident at Harvard Medical School's main psychiatric teaching facility, the Massachusetts Mental Health Center.

The public's so-called "mistaken" image of ECT is, in reality, based on modern modified ECT, which has been around for a long time. As mentioned earlier, it is actually more dangerous than the older forms. The electrical currents must be more intense to overcome the anticonvulsant effects of the sedatives that are given during modified ECT (Breggin, 1979). Too frequently, the patient is routinely given a sleeping medication or tranquilizer the night before, further increasing the brain's resistance to having a seizure. Although ECT experts recommend against it, commonly patients are prescribed multiple psychiatric drugs at the same time. In addition, patients are exposed to the added risk of anesthesia. Other modifications include changes in the type of electrical energy employed and the use of unilateral shocks applied to the nondominant (nonverbal) side of the brain. However, the efficacy of these modifications remains controversial among shock advocates and, as a result, older methods continue to be used much or even most of the time (Sackeim et al., 2007).

Since the APA (1990b) task force does not exclusively endorse the modified forms of ECT, the claim that modern ECT is somehow much safer is again undercut. Besides, as already emphasized, some ECT advocates give excessive doses—beyond the dose required to produce a convulsion. Sackeim has advocated using electrical doses so large that the safety controls on the machines have to be disabled (Sackeim et al., 1993).

There is no reason to believe that shocking the nonverbal side of the brain is less harmful. As Blakeslie (1983) confirmed, damage and

dysfunction on the nonverbal side are more difficult for the individual to recognize or describe (see discussion of anosognosia in chapter 1). But the defects are no less devastating. Injury to the nonverbal side impairs visual memory, spatial relations, musical and artistic abilities, judgment, insight, intuition, and personality. Because of the victim's difficulty in perceiving damage to the nondominant side of the brain, and because it impairs judgment and insight, modified nondominant ECT is probably more spellbinding. Meanwhile, it is ironic that biopsychiatry promotes sacrificing the nonverbal side of the brain, while humanistic psychology is emphasizing its importance to the full development of human potential.

The Brain-Disabling Principle

Beginning with Cerletti and Bini, who introduced electroshock in 1938 in Italy, many advocates of the treatment have *not* wanted to make the treatment less harmful to the brain. They have considered brain damage necessary for the cure and often spoke openly about it (Cerletti, 1940; reviewed in Breggin, 1979).

Fink, himself a member of the 1978 and 1990 APA ECT task forces, for decades argued and demonstrated scientifically that ECT's "therapeutic" effect is produced by brain dysfunction and damage. He pointed out in his 1979 textbook that "patients become more compliant and acquiescent with treatment" (p. 139). He connected the so-called improvement with "denial," "disorientation" (p. 165), and other signs of traumatic brain injury and an organic brain syndrome. This is a direct confirmation of the brain-disabling treatment and the use of iatrogenic denial in authoritarian psychiatry.

Fink was even more explicit in earlier studies. In 1957, he stated that the basis for improvement from ECT is "craniocerebral trauma." In 1966, Fink cited research indicating that after ECT, "the behavioral changes related to the degree of induced trauma" (p. 475). Referring to the multiple abnormalities produced in the brain following ECT, he wrote, "In these regards, induced convulsions in man are more similar to cerebral trauma than to spontaneous seizures" (p. 481). He stated that improvement depends on the development of an abnormal EEG and other changes in the brain and spinal fluid typical of trauma and compared ECT to "cerebral trauma" (p. 48). Fink (1966) cited Tower and McEachern (1949), correctly stating that they "concluded that spinal fluid changes in induced convulsions were more like those of craniocerebral trauma than those of spontaneous epilepsy." He then gave further evidence for this comparison between ECT and traumatic brain injury.

Up to at least 1974, Fink continued to propose that ECT has its effect by traumatizing or damaging the brain. He began his discussion by

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noting that psychiatric treatments have often been “drastic” and then cited, among other examples, heat and burning, bleeding, water immersion, and craniotomy. He then went on to present several axioms of ECT, including the connection between the supposed therapeutic effect and traumatic changes in the brain. He spoke directly of the producing “cerebral ‘trauma’” (p. 9) reflected in EEG slow wave activity. He compared induced convulsions to “craniocerebral trauma” (p. 10). He attributed improvement to the increased use of “denial” by the patient and to the development of “hypomania” (p. 14)—both clinical signs of profound irrationality caused by brain damage and dysfunction.

Psychiatry’s more recent emphasis on proving that ECT is harmless has developed in response to scientific criticism of the damaging effects made by me and by others, such as neurologist John Friedberg (1976, 1977) and shock survivor Leonard Frank (1979, 1980, 1990, 1991, 2001). Thus, the APA (1990b) task force report, despite Fink’s participation, made no such comparisons between head injury and ECT; instead, the report dismissed any suggestion that the treatment is severely traumatic. In depositions and trial testimony in defense of doctors who give ECT, Fink now takes the position that ECT causes no brain damage.

The 1990 APA task force report noted that low-dose unilateral ECT is often less effective than forms of ECT that deliver more electrical energy. This observation tends to confirm the brain-disabling principle that so-called therapeutic efficacy is a function of the degree of treatment-induced damage.

Sackeim et al. (1993) covertly revived the concept promoted by ECT pioneers that a therapeutic response depends on inflicting brain damage and dysfunction. They advocated bilateral ECT—the most obviously damaging method—using a dose of electricity 2.5 times that required to induce a convulsion in the patient. I evaluated a case in which a doctor followed Sackeim et al.’s published recommendation and gave his patient the increased dosage. The patient suffered severe, irreversible memory loss and chronic mental dysfunction, rendering her permanently unable to work at her previously high intellectual level.

Psychiatric drugs are nowadays frequently justified on the grounds that they correct biochemical imbalances. Like Prozac, shock treatment is said to work by enhancing serotonin (e.g., Abrams, 1988). Accepting this rationale requires ignoring the more gross damage being done: The shocked brain is so traumatized that the patient is rendered too confused and blunted to feel any subtle emotions. Even psychosurgery is nowadays sometimes justified on the grounds that it corrects biochemical imbalances. One advocate looks forward to delivering serotonin “psychosurgically” to “serotonin-depleted sites” in the brain (Rodgers, 1992, p. 106).

Iatrogenic Helplessness and Denial, and Spellbinding

ECT provides a prototype for the concept of iatrogenic helplessness and denial, and spellbinding (chapter 1). Controlled studies of ECT show that any therapeutic effect evaporates after 4 weeks—the approximate time it takes to recover from the most severe symptoms of organic brain syndrome or delirium. Except for psychosurgery, ECT provides the most extreme example in which the psychiatrist denies the damage he is doing to the patient, and then utilizes the effects of that damage to produce a less emotionally aware, less autonomous, and more manageable patient. As Max Fink's earlier work openly described, through brain damage and the exercise of medical authority, patients are pushed deep into denial about the harm done to them as well as about their still unresolved personal problems. This is an example of profound spellbinding intentionally inflicted on the patient under the guise of treatment.

Consistent with other victims of central nervous system damage, most ECT patients minimize or deny their real losses of mental function. This denial of mental dysfunction in brain-damaged patients is called anosognosia (discussed in chapter 1). While damage to either side of the brain can produce anosognosia, it seems more common following damage to the nondominant side (in right-handed individuals, the right is usually nondominant). In electroshock treatment, at least one electrode lies over the nondominant side. In contemporary ECT, both electrodes are frequently placed over the nondominant side. As already noted, damage to the nondominant side of the brain impairs judgment and insight without the patient realizing it, making the treatment very spellbinding.

Nondominant shock starkly illustrates the principle of iatrogenic helplessness and denial: The doctor damages the brain in such a way as to confound the patient's ability to perceive the resulting dysfunction.

Advocates of ECT are well aware that shock patients suffer from anosognosia and denial and therefore cannot fully report the extent of their memory losses and mental dysfunction. Yet these same advocates claim that patients exaggerate their post-ECT problems.

Interviews with family and friends of patients often disclose that they are painfully aware of the damage done to their loved ones. Often, the psychiatrist is the only one who consistently and unequivocally denies the patient's damaged state.

A LONG CONTROVERSY SURROUNDING ECT

The 1978 APA task force report labeled electroshock treatment as controversial. The 1985 Consensus Conference on ECT report stated,

"Electroconvulsive therapy is the most controversial treatment in psychiatry" and referred to 45 years of dispute surrounding issues such as efficacy and "possible complications." In the opening sentence of the introduction to Abrams's (1988) book, Fink referred to the "more than 50 years of controversy" surrounding ECT.

Since my 1979 book, I have hammered at the right of patients to know that ECT is a controversial treatment, and I have cited the previous quotations in medical-legal reports and testimony. Many survivors of shock treatment, such as David Oaks of MindFreedom and Leonard Frank, have made similar points. Perhaps as a result, the 1990 APA task force report said not a word about controversy. ECT is presented as if no one in the profession has ever criticized it. Psychosurgery remains the only treatment surrounded by more controversy than ECT, but it is used much less frequently (Breggin et al., 1994b). The two treatments are closely related in many ways. Electroshock can be understood as "closed-head electrical lobotomy."

The most significant challenge to ECT within the medical profession was launched by neurologist John Friedberg (1976), whose book for laypersons was followed by a journal review (Friedberg, 1977). Friedberg's publications were quickly followed by a volume edited by Leonard Frank (1978) and a book by this writer (Breggin, 1979). Reviews of ECT-induced damage to the brain and mind have continued to be published in professional journals (Cameron, 1994; Frank, 1990; Templer, 1992). Templer and Veleber (1982), for example, summarized their review of the literature:

Some human and animal autopsies reveal permanent brain pathology. Some patients have persisting spontaneous seizures after having received ECT. Patients having received many ECTs score lower than control patients on psychological tests of organicity, even when degree of psychosis is controlled for.

A convergence of evidence indicates the importance of the number of ECTs.... Our position remains that ECT has caused and can cause permanent brain pathology.

Boyle (1986) reviewed the literature and stated,

In conclusion, there is considerable empirical evidence that ECT induces significant and to some extent lasting brain impairment. The studies cited above are but a few which suggest that ECT is potentially a harmful procedure, as indeed are most naturally occurring episodes of brain trauma resulting in concussion, unconsciousness and grand mal epileptic seizures. Accordingly, the continued use of ECT in psychiatry must be questioned very seriously. (p. 23)

After hearing evidence presented to the Food and Drug Administration's Respiratory and Nervous System Device Panel, consumer representative Susan Bartlett Foote (1983) reported back to the FDA that

evidence of the safety and efficacy of ECT devices remains controversial and conflicting. The "new evidence" submitted [by the American Psychiatric Association] petition did not, by any means, eliminate the unanswered or troubling questions surrounding safety and efficacy of the machines. (p. 2)

Consider that all of this was published before Sackeim et al.'s (2007) study showing permanent harm to the brain and mind caused by ECT. Psychiatry has ignored the decades of research that long ago should have brought the treatment to a halt.

Survivors of shock treatment have become an increasingly active force. In addition to writing and appearing in the media, many who have undergone ECT continue to protest at national psychiatric conventions and shock symposia and even chain themselves to the gates and doors of so-called "shock mills."

More than 30 states have passed legislation to monitor ECT, set limits on the number of treatments or the age at which it can be given, and require second opinions and informed consent. Four states have banned its use on children, most recently Texas. While efforts to require informed consent have proved almost impossible to enforce in the face of psychiatric resistance, they have raised further questions about the use of shock treatment. However, critics of shock have relatively little clout or funding compared with the American Psychiatric Association and organized shock advocates, who have fought continuously against any monitoring or any restraint of ECT; little progress in reform has been made in recent years.

The most dramatic threat to shock treatment became known as the "Berkeley ban." Ted Chabasinski, who had been subjected to electroshock as a child, organized a grassroots citizens' movement in support of a referendum to ban ECT in Berkeley, California. After the proposition was overwhelmingly approved by the electorate, the psychiatric establishment, led by the APA, intervened and had the ban overturned in court. But the survivors could claim a partial victory—a so-called "power outage" of 41 days at Herrick Hospital, the city's only ECT facility, in the winter of 1982.

California again became the center of public criticism of electroshock. Inspired by a coalition of former patients and concerned professionals, Angela Alioto, a member of the San Francisco Board of Supervisors, held hearings on ECT. About two dozen "shock survivors" testified about

permanent damage to their brains and minds. Although both sides had ample time to organize, no shock patients showed up to offer testimonials in favor of the treatment (Breggin, 1991b, 1991c; Frank, 1991).

The recommendations of Alioto's committee were adopted by the city's governing body and signed by Mayor Art Agnos on February 20, 1990. The resolution declared the opposition of the Board of Supervisors to the "use and financing" of ECT in San Francisco (Figueroa, 1991). It also called for the state legislature to develop more strict requirements for informed consent, including the exposure of potential patients to live or videotaped presentations by critics of the treatment. The resolution, which followed the recommendations made in my testimony at the Alioto hearings, was not legally binding. While the resolution has been an important moral and educational victory for electroshock opponents, its actual impact was negligible.

David Oaks is the executive director of MindFreedom (<http://www.mindfreedom.org>), the leading survivor organization in the world fighting for psychiatric patient rights and resisting psychiatric abuses. He edits the group's magazine, organizes protests against psychiatric abuses like electroshock treatment, and in general inspires reform-minded professionals and victims alike.

THE NEED TO BAN ECT

The 1990 APA task force report represented a disillusioning and disappointing watershed for my own reform activities around ECT. I had long argued that ECT was an ineffective, dangerous, anachronistic treatment that should be abandoned by modern psychiatry. Yet, despite the urging of many victims of ECT, I refused for many years to endorse public or legislative efforts to ban it. It was my position that the practice of medicine and the rights of patients were better served by insisting on informed consent—and by holding liable those psychiatrists who fail to convey to their patients the controversial nature of ECT and its potentially damaging effects. Unfortunately, the 1990 APA report and the APA's political pressuring of the FDA demonstrated that organized psychiatry was determined not to inform professionals or patients about the risk of ECT. Despite the disclaimer tucked away on its copyright page, the APA report provided a shield for those who recommend and administer ECT—an "official" conclusion that there is no serious risk of harm. Doctors who prescribe or recommend ECT can try to hide behind this report when their injured patients protest to them or bring legal actions.

In the environment created by the APA, informed consent for ECT became a mirage. Therefore, after much initial hesitation, I decided to

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endorse public efforts to ban ECT. I believe that all concerned mental health professionals should support the banning of ECT.

Given that even the APA and the FDA published fraudulent claims about the harmlessness of ECT, it is fair to conclude that patients are rarely if ever going to be given informed consent by doctors who advocate the treatment. Because ECT promoters like Max Fink, Richard Abrams, and Harold Sackeim are considered believable authorities by their colleagues, practicing psychiatrists feel safe in telling their patients that ECT is relatively harmless and very effective.

I have read sworn testimony by many shock doctors, reviewed the medical charts of their patients, and seen the "consent" forms that they give to their patients—and I have never seen a case in which a patient was given adequate information about the treatment's brain-damaging effects. If they were informed about the results of animal experiments or the results of Sackeim et al.'s (2007) recent research, all but the most self-destructive patients would refuse the treatment. Because ECT patients will never be given informed consent, the only alternative is a ban on the treatment. Some patients do feel "helped" by ECT. Often, they have been so damaged that they cannot judge their own conditions. They suffer from ECT spellbinding, as well as iatrogenic denial and helplessness. But should a treatment be banned when some people believe they are helped by it? In fact, it is commonplace in medicine and psychiatry to withdraw treatments and devices that have caused serious harm to a small percentage of people, even though they may have helped a very large percentage. The risk of serious injury to a few outweighs helping many. In the case of ECT, a large percentage of people are being harmed, and there is little evidence that any are being helped.

CONCLUSION

Based on the original large-animal studies that demonstrated ECT-induced brain damage, organized psychiatry should have banned the "treatment" decades ago. Even without the animal studies, Sackeim et al.'s (2007) demonstration of permanent ECT-induced memory loss and other cognitive deficits consistent with dementia should have been sufficient to stop all use of the treatment. This chapter has also reviewed a mountain of additional research confirming that ECT damages both the brain and the mind.

There is no need to advocate for additional research. Why damage the brains of more animals and more people? The facts have been conclusively established. Shock treatment physically damages the brain, irreversibly impairs mental function, and ruins the lives of many if not most

patients who are subjected to it. On top of that, controlled clinical trials comparing ECT to sham ECT show no advantage to the treatment. ECT should be utterly discarded as a useless, damaging relic from psychiatry's more violent past.

Unfortunately, psychiatry shows not the slightest inclination to rein in its compulsion to damage the brains of its patients in the name of "treatment." Sackeim et al.'s (2007) study aroused no concern whatsoever within the profession. Psychiatry's more abusive treatments, such as ECT, will never be stopped by psychiatry itself. ECT will have to be stopped by forces outside the profession including public outrage, court decisions prohibiting its use, and legislation banning it.

NOTE

1. Devanand is one of the authors in Sackeim et al. (1993) calling for the use of intensive electroshock using 2.5 times the electrical current required to produce a convulsion.

EXHIBIT 35

Uncommon but Serious Complications Associated With Electroconvulsive Therapy: Recognition and Management for the Clinician

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Electroconvulsive therapy (ECT) is a safe and effective treatment for severe mood disorders. Rarely there can be serious complications, such as postictal agitation, cardiovascular compromise, prolonged seizures, and status epilepticus, all of which are important for the clinician to recognize and treat. Postictal agitation can be severe, requiring emergent intervention and subsequent prophylactic measures to avoid premature ECT discontinuation. Cardiovascular responses to ECT include significant hemodynamic changes that may result in complications, even in patients without preexisting cardiovascular conditions. However, preexisting cardiovascular conditions per se are not contraindications to ECT in patients with disabling psychiatric disease. Recognizing and treating prolonged seizures is essential to prevent progression to status epilepticus. Failure to recognize and treat any of these events may result in increased mortality and morbidity. Understanding such complications and their management strategies avoids unnecessary treatment discontinuation due to manageable ECT complications.

Introduction

Electroconvulsive therapy (ECT) is recognized as a highly effective and safe treatment in psychiatry. Despite sometimes being used in older adult patients with concomitant medical problems, mortality from the procedure is low (~ 1–2 cases per 100,000 ECT sessions). This would be

expected to translate to about 1 death for every 10,000 patients treated. From a morbidity standpoint, ECT is also quite well tolerated. The most common side effects are headache, muscle aches, transient confusion, and memory difficulties, with the latter usually mild and transient. Nevertheless, more serious complications, such as postictal agitation (PIA), arrhythmias, myocardial infarction, prolonged seizures, and status epilepticus (SE), occur occasionally. All clinicians who encounter patients undergoing ECT must be vigilant for such complications so that they can be recognized promptly and steps can be taken to treat them appropriately and prevent recurrence during future ECT sessions.

Postictal Agitation

PIA is a relatively frequent clinical complication with ECT, reported in up to 7% of ECT sessions [1] and in 10% of patients who undergo an ECT course [2]. Clinical characteristics of agitation in the setting of ECT include disorientation, motor restlessness, aimless movements, nonresponse to verbal commands, panic-like behavior or fear, and even attempts to leave the recovery room [1,3•]. An episode can last from 5 minutes to 1 hour or more [4]. It is important for the clinician to keep in mind that what initially seems like agitation may be nonconvulsive epileptic activity. Therefore, the electroencephalography (EEG) trace needs to be assessed promptly to rule out prolonged seizure as a cause of PIA [5].

Etiology

Although this entity is often idiopathic in origin [3•], specific factors such as electrode placement, concomitant lithium use, dosing of the anesthetic induction agent and muscle relaxant, and high pre-ECT anxiety levels all have been implicated in PIA onset [4,6]. Episodes of PIA occur on a spectrum of severity and range from mild and self-limiting to very severe. Very severe episodes require

emergent interventions to control the acute situation along with subsequent prophylactic measures at future sessions so as to avoid premature ECT discontinuation [7,8].

Management

Nonpharmacologic interventions such as the brief use of restraints and maintenance of intravenous (IV) access are primary measures to ensure patient safety and permit efficient pharmacologic treatment. As stated previously, the first action to be taken when a patient manifests PIA is to examine the EEG to determine whether the problem is one of prolonged seizure. If this is the case or if in doubt, IV antiseizure medication needs to be administered without delay. Either way, emergent management of PIA with short-acting benzodiazepines such as midazolam and lorazepam represents a first-line intervention that can serve both purposes [4,9]. A good second-line option is haloperidol, 5 to 20 mg IV [3•]. Propofol boluses of 0.1 to 2.0 mg/kg repeated every 3 to 5 minutes, followed by infusion at 25 to 150 µg/kg per minute are effective for resistant cases of PIA, as illustrated by a recent case series of severe PIA from our center. Methohexital boluses also can be effective for severe PIA with the advantage of being immediately at hand when methohexital was used as the anesthetic agent [10]. An effective bolus is usually half the dose used for induction of anesthesia.

Prophylaxis

Addressing psychiatric factors

The best prophylaxis for PIA is to treat the cause, if one can be identified. Concomitant lithium therapy should be discontinued or at minimum dosing held for 24 hours before each ECT session, a strategy that has been shown to reduce PIA incidence [10]. Bilateral electrode placement is more commonly associated with agitation and may subside when switching to a unilateral placement, if this is clinically feasible [11]. Similarly, strongly left-handed patients presenting with agitation after receiving right unilateral ECT may improve when switched to a contralateral placement so as to stimulate the patient's nondominant hemisphere in this instance [11].

Addressing anesthetic factors

Anesthetic contributory factors also may need to be addressed. Insufficient dosing of induction agents and muscle relaxants has been linked to PIA. Post-ECT agitation has been prevented in some cases by increasing the dose of the anesthetic induction agent methohexital to about 1.1 mg/kg and/or adding a bolus of about 0.7 mg/kg immediately at seizure end [12,13]. Increasing the dose of the muscle relaxant agent succinylcholine also has been used to reduce PIA occurrence [1]. It has been hypothesized that raising the dose of succinylcholine may help with PIA by reducing release of lactate from muscle through minimization of muscular activity

during ECT. Elevated lactate levels have been linked to PIA [1]. Propofol in boluses of 0.1 to 2.0 mg/kg initially after seizure, followed by an infusion if needed (25–150 µg/kg per minute) has been shown to be an effective intervention when other pharmacologic therapies have been unsuccessful in managing this clinical problem [3•]. An oral alternative to these intravenous medications, promethazine, 25 to 50 mg given orally 2 hours before each ECT session, also has been proposed for prophylaxis of PIA [8].

Case example

A 71-year-old male patient underwent ECT treatment of his refractory major depressive disorder. The course was successfully administered except for the presence of PIA after session number five. That episode was controlled by lorazepam and haloperidol. Subsequent severe episodes of PIA were resistant to benzodiazepines and antipsychotics in combination. Propofol administration was required to resolve the agitation spells. Propofol was started as a prophylactic measure at session eight, and the patient successfully completed the ECT course without recurrence of PIA.

As this case illustrates, emergence of severe PIA does not mandate cessation of the ECT course. Physicians administering ECT and recovery room nurses should be aware of this relatively frequent complication and of the range of strategies for its acute and prophylactic treatment.

Cardiovascular Complications of Electroconvulsive Therapy

Acute cardiovascular responses to ECT involve the sympathetic and parasympathetic branches of the autonomic nervous system. These result in transient but quite significant hemodynamic changes, including an initial period of bradycardia [14], followed by a catecholamine surge with increases in blood pressure and heart rate that ordinarily resolve over several minutes after the seizure has ended [15]. Studies have shown the rate of ECT-associated cardiovascular complications to be 7.5% in patients without preexisting cardiovascular conditions, generally in the form of benign poststimulation transitory arrhythmias that resolved within minutes [16]. Studies have reported a much higher rate (55%) of ECT-associated cardiovascular complications in patients with preexisting cardiovascular conditions [16]. Fortunately, most of these events also correspond to transitory arrhythmias that are self-correcting or controlled using β blockers such as esmolol (a short-acting selective β_1 blocker) and labetalol (a nonselective β blocker and α_1 blocker), which appear to attenuate effectively these acute hemodynamic responses, even in the setting of preexisting cardiovascular conditions [15,16]. In addition, according to one review, as many as 91% of these patients were able to safely complete their ECT courses [16].

Risk/Benefit

Although transient in nature, hemodynamic responses to ECT may have potential in some susceptible patients to lead to more serious outcomes, such as myocardial infarction, circulatory collapse, and atrial and ventricular arrhythmias [17,18]. This can make the decision of whether to administer ECT to a patient with preexisting cardiovascular problems challenging. However, some older adult patients in particular already may have been unable to tolerate antidepressants at an effective dose, making ECT the most feasible biological treatment [16]. Risks of administering ECT in such instances also should be weighed against the risks associated with untreated severe mood disorders, including suicide [19]. In terms of risks of proceeding with ECT in such circumstances, it is reassuring to note that it has been safely and successfully administered in the setting of significant cardiovascular morbidity (eg, pulmonary hypertension, cerebral aneurysm, severe aortic stenosis, recent and active myocardial infarction, post-aortic aneurysm surgery) and in patients with existing abdominal aortic aneurysm [14,19].

Pre-electroconvulsive therapy risk assessment

Careful consultation with cardiology and anesthesiology services is warranted in each case to assess risk and for preprocedure planning and interventions so as to minimize risk of ECT-related complications [20•].

Case example

Aortic stenosis

The following case, which we recently reported, illustrates that the risk/benefit profile may still be favorable in older adult patients with underlying cardiovascular issues and needs to be weighed on a case-by-case basis [20•].

A 96-year-old female patient with severe aortic stenosis experienced a relapse of her severe depression when ECT had been withheld due to increased concerns regarding medical risks of ECT given her advanced age and the degree of aortic stenosis. With relapse of depression, the risk/benefit of continuing with ECT was reappraised. Reassessment of the case by cardiologists at our institution confirmed severe stenosis with a valve area of 0.5 cm² and a peak pressure gradient across the valve of 110 mm Hg. However, the ventricular ejection was normal at 70%, and after a careful weighing of the risk of ECT treatment versus the risk of withholding ECT, it was decided to proceed with ECT in this instance. In the event, ECT was very well tolerated by the patient, and she experienced a full remission of symptoms. She continues to receive successful monthly maintenance ECT without evidence of any cardiovascular complications secondary to ECT.

Takotsubo Cardiomyopathy

A different clinical scenario presents itself when a new cardiovascular issue develops as a complication of ECT

during the acute course. We recently reported the first published case of Takotsubo cardiomyopathy (TCM) as an unexpected cardiac complication associated with ECT [21•]. TCM is a reversible cardiomyopathy initially indistinguishable from a myocardial infarction that appears to be precipitated by an acute stressor and the related catecholamine surge [22,23]. This surge appears to cause a stunning of the myocardium, producing a reversible cardiomyopathy. ECT produces a similar catecholamine surge [24] and therefore may carry a risk of inducing TCM.

Clinically, TCM manifests as a syndrome of chest pain, dyspnea, ST-segment elevation on electrocardiogram, and elevated cardiac enzyme levels in the absence of evidence of an obstructive lesion on coronary angiography [25]. Characteristic echocardiographic findings include hypokinesis of the left ventricle's apical segments [26].

Case example

A 45-year-old female nonsmoker with a negative family history for cardiac disease and no evidence of coronary artery disease underwent a course of ECT for treatment-resistant major depression. Normal electrocardiogram and routine laboratory values and standard clearance by medicine and anesthesiology were obtained before ECT. The first two sessions were successfully administered without any reported complication. After session three, the patient reported substernal chest pain that subsided spontaneously after 15 minutes. Despite a normal electrocardiogram, cardiac enzymes were found to be elevated, confirming a diagnosis of acute myocardial infarction. Cardiac work-up to determine etiology showed no evidence of coronary artery disease on catheterization. Echocardiogram and CT findings were consistent with a diagnosis of TCM. Over a few days, the cardiomyopathy resolved, and echocardiographic findings normalized. It was decided in this instance to discontinue the course of ECT, and treatment of her depression reverted back to pharmacotherapy.

As this case illustrates, significant cardiovascular complications may occur rarely in association with ECT, even in patients without cardiovascular risk factors or comorbidities. Transient chest pain after ECT, even in the absence of electrocardiogram abnormalities, should be investigated to rule out cardiovascular conditions that warrant ECT discontinuation or modification to avoid untoward sequelae for the patient [21•].

Prolonged Seizures

A prolonged seizure is defined as a seizure lasting longer than 3 minutes by motor or electrocardiogram manifestations [27–29]. Prolonged seizure incidence ranges from 1% to 2% during ECT [30]. Adolescents are reported to have a higher frequency of prolonged seizures [31]. Medications such as theophylline, lithium, caffeine, or trazodone and medical conditions that lower seizure threshold,

such as electrolyte disturbances, have been associated with increased risk of prolonged seizures [27–29]. Routine use of simultaneous electrocardiogram monitoring has improved identification of prolonged seizures during ECT. Prolonged seizures after ECT may result in increased postictal confusion and amnesia [27,32]. Early recognition and treatment are particularly important for patients receiving maintenance ECT on an outpatient basis to prevent progression of prolonged seizures into SE or nonconvulsive SE (NCSE) with resultant increase in morbidity and mortality.

Treatment

Prolonged seizures can be terminated by IV administration of a short-acting benzodiazepine (eg, midazolam, 1–2 mg) or a repeat dose of the anesthetic agent (eg, methohexital) [33]. The dose should be repeated within 2 minutes if the seizure activity continues. Lorazepam, 2 to 4 mg IV over 1 minute, or diazepam, 5 to 10 mg IV, may be given for continued epileptic activity [33]. It is important to maintain oxygenation and closely monitor vital signs, including electrocardiogram. If the seizure activity persists after lorazepam or diazepam administration, an SE algorithm should be followed and a neurology consultation obtained expeditiously [33].

Status Epilepticus

SE is a life-threatening emergency that is defined by the International League Against Epilepsy as prolonged epileptic seizure activity that persists for a sufficient length of time or is repeated frequently enough to produce a fixed and enduring epileptic condition [34]. SE results in increased mortality (up to 22% in refractory cases) and morbidity (eg, hyperthermia, circulatory collapse, and hypoxic encephalopathy) [35]. The pathophysiology of SE is described as failure of the biochemical inhibitor mechanisms to terminate an isolated seizure and persistence of low seizure threshold despite repeated seizures [36].

Etiology

Convulsive and nonconvulsive forms of SE have been reported as complications of ECT since its introduction [37]. With the routine application of general anesthesia and muscle relaxation during ECT, SE incidence has been reduced [38]. Hypoxia, medications that lower seizure threshold, hyperoxygenation, and cerebral hyperexcitability are associated with increased risk of SE [37,39]. Patients with a history of epilepsy have been reported to be at slightly increased risk for prolonged or spontaneous seizures during ECT. However, ECT also may be associated with improved seizure control in epilepsy patients due to an increase in seizure threshold [29,30,38]. SE also has been reported after ECT in a pregnant patient [12]. Pregnancy may alter the seizure threshold through hormonal changes, sleep deprivation, and electrolyte disturbances

[39]. SE has been reported with unilateral and bilateral ECT. It occurs more commonly when several seizures are induced during one period of anesthesia [37,39].

Recognition and treatment of status epilepticus

Electrocardiogram monitoring is important in confirming the diagnosis, particularly in the absence of convulsive activity or presence of agitation or other motor behavior abnormalities that can interfere with the diagnosis of epileptic activity. Treatment of SE should be started within 10 minutes of onset, as seizure activity that persists longer than 30 minutes results in greater morbidity and mortality [40,41]. Delayed treatment also has been associated with poor treatment response due to alterations in γ -aminobutyric acid type A receptors, resulting in decreased sensitivity to benzodiazepine treatment [41].

Lorazepam has been demonstrated to be more efficacious than phenobarbital, phenytoin, or a combination of phenytoin and diazepam in the Status Epilepticus Cooperative Study Group trial [42]. Thus, lorazepam, 0.1 mg/kg, is widely used for rapid seizure control with addition of phenytoin, 18 to 20 mg/kg, to sustain seizure control. Phenobarbital, 20 mg/kg, is often used as a second-line treatment with assisted ventilation. In refractory cases, when SE fails to respond to lorazepam, phenytoin, or phenobarbital, sedation with IV propofol or midazolam with close monitoring in an intensive care unit is required [40,41].

Prolonged seizures or SE do not absolutely preclude further ECT. Consultation with neurology and prophylaxis with anticonvulsants may be required to prevent future SE episodes after ECT [43].

Nonconvulsive status epilepticus

Case example

A 78-year-old woman with bipolar disorder type I without a past medical history of seizure disorder was successfully maintained on weekly outpatient ECT after an episode of severe major depression with psychotic features. On the morning of her 12th treatment, the patient was noted to have word-finding difficulty, short-term memory impairment, and disorientation. Neurologic examination was within normal limits, except for significant expressive aphasia. MRI of the brain showed mild generalized cerebral atrophy. An EEG revealed left temporal epileptic activity consistent with a complex partial SE also supported by the clinical findings. The patient was started on lorazepam and phenytoin with near-complete resolution of epileptic activity on repeat EEG within 7 days of initial symptom onset.

NCSE is defined as a form of SE that occurs in the absence of motor manifestations [34]. Although NCSE does not result in the systemic complications of convulsive SE, early recognition and treatment of this rare complication are important to prevent neurologic sequelae [37]. Several authors have reported NCSE after ECT as a rare

complication of ECT [32,37,43,44]. NCSE can complicate ECT at any age, ranging between 18 and 87 years of age in different case reports [32,37,43,44]. There does not seem to be a gender predilection [10]. NCSE has been observed after the first ECT session or as late as the 13th treatment, lasting 2 hours to 5 days, most commonly in the form of absence seizures [32,44]. NCSE has been reported after both unilateral and bilateral ECT. Risk factors for NCSE development include a history of epilepsy, structural brain lesions, and medications that lower seizure threshold.

Recognition and treatment

NCSE is commonly unrecognized or misdiagnosed [37,45]. Acute, unexplained changes in mental status or behavior after ECT are the hallmark findings of NCSE. The clinical features are often subtle and can also affect memory and other domains of cognition [37,45]. Alterations in muscle tone and fine facial or limb tremors can be observed on neurologic examination in complex partial form of NCSE [45]. Differential diagnosis also should include other clinical states that can contribute to an acute change in mental status or behavior, such as post-ECT delirium or agitation, catatonia, or exacerbation of a manic or psychotic episode. Mild confusional states after ECT are relatively common; however, they are typically short-lived and resolve spontaneously without specific interventions. Routine blood chemistry should be obtained to rule out electrolyte disturbances. Neuroimaging is also important to rule out acute intracranial ischemic or hemorrhagic events, especially in geriatric patients. EEG monitoring is the gold standard diagnostic instrument for identifying NCSE in the absence of convulsions. Detection of seizure activity on EEG confirms the diagnosis in most cases [46]. Normalizing EEG activity on repeat EEG monitoring after treatment with benzodiazepines also supports a diagnosis of NCSE, in addition to clinical response [46]. EEG recordings after ECT may show marked nonspecific alterations that could mask but also resemble seizure activity [46,47]. Fink [47] has made the counterargument that the slowing of EEG frequencies and the recording of seizure-like activity are routinely observed in the interictal recordings in the absence of motor symptoms and that these recordings can be misinterpreted as NCSE.

Benzodiazepines are the treatment of choice for NCSE [37,44,45]. The treatment guidelines for convulsive SE also apply to NCSE, as detailed previously. Benzodiazepines often lead to a rapid and dramatic improvement in clinical and EEG findings [45].

Electroconvulsive Therapy as a Treatment Modality for Status Epilepticus

ECT also has been used to treat medication-refractory SE [41,48–50]. SE has been conceptualized as persisting seizure due to a failure of inhibitory mechanisms that terminate a seizure activity [42]. However, seizures

produced by ECT mimic generalized tonic clonic seizures and activate the inhibitory mechanisms needed to abort a seizure, unlike the failure of inhibition in SE [41,50]. Enhanced γ -aminobutyric acid transmission has been suggested as a possible mechanism for anticonvulsant effects of ECT [48–50]. Animal and human studies have shown that ECT results in an increased seizure threshold, typically with successive courses of treatment [28,48]. For better neurologic outcomes, it is important to consider ECT as a treatment option for refractory SE before irreversible brain damage ensues [41]. The American Psychiatric Association Task Force Report on ECT has listed refractory epilepsy and SE as diagnostic indications for ECT [29]. Based on case reports, it has been suggested that the frequency (eg, three ECT sessions per day) and intensity of ECT needed to treat SE may be greater than those required for major depression [49].

Conclusions

After 70 years of clinical use, ECT remains a gold standard treatment in psychiatry for the most difficult to treat mood disorders, particularly bipolar and unipolar major depressive episodes that do not respond to standard pharmacotherapies. It can also be a life-saving intervention for catatonia. The risk/benefit is generally very favorable for ECT, even in cases of older adult patients who have significant concomitant medical disorders. Its only absolute medical contraindications are raised intracranial pressure and in the immediate aftermath of a myocardial infarction. Nevertheless, like any medical procedure requiring administration of a general anesthetic, serious complications can occur.

PIA is readily recognized and requires acute intervention for the safety of the patient and staff and future prophylaxis to make possible further ECT administration if clinically warranted. Transient hemodynamic changes are expected with ECT; their management is part of standard operating procedures during ECT. More subtle entities, such as a silent myocardial infarction or TCM, can be easily missed and require significant vigilance to be recognized quickly. Lastly, prolonged seizures and status can occur with ECT and must be dealt with promptly and vigorously. However, ironically, ECT also has been successfully used to treat refractory SE.

Disclosures

Dr. O'Reardon has received grant support from Bristol-Myers Squibb, Cyberonics, Eli Lilly and Company, the Magstim Company, Neuronetics, Pfizer, and Sanofi-Aventis; has served as a consultant for Eli Lilly and Company and Neuronetics; and is a member of the speakers' bureau for Eli Lilly and Company and Bristol-Myers Squibb. No other conflicts of interest relevant to this article were reported.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Auriacombe M, Reneric JP, Usandizaga D, et al.: Post-ECT agitation and plasma lactate concentrations. *J ECT* 2000, 16:263–267.
2. Swartz CM: Electroconvulsive therapy emergence agitation and succinylcholine dose. *J Nerv Ment Dis* 1990, 178:455–457.
3. O'Reardon JP, Takieddine N, Datto CJ, Augoustides JG: Propofol for the management of emergence agitation after electroconvulsive therapy: review of a case series. *J ECT* 2006, 22:247–252.
- A 10-patient case series that suggests that propofol represents a new and very effective treatment option for severe treatment-resistant PIA.
4. Augoustides JG, Greenblatt E, Abbas MA, et al.: Clinical approach to agitation after electroconvulsive therapy: a case report and literature review. *J ECT* 2002, 18:213–217.
5. Trimble MR, Ring HA, Schmitz B: Neuropsychiatric aspects of epilepsy. In *Neuropsychiatry*. Edited by Fogel BS, Schiffer RB. Baltimore: Williams and Wilkins; 1996:771–803.
6. Gomez J, Dally P: Intravenous tranquilization with ECT. *Br J Psychiatry* 1975, 127:604–608.
7. Logan C, Stewart J: Treatment of post electroconvulsive therapy delirium and agitation with donepezil. *J ECT* 2007, 23:28–29.
8. Vishne T, Amiaz R, Grunhaus L: Promethazine for the treatment of agitation after electroconvulsive therapy. *J ECT* 2005, 21:118–121.
9. Labbate LA, Miller JP: Midazolam for treatment of agitation after ECT. *Am J Psychiatry* 1995, 152:472–473.
10. Small JG, Milstein V: Lithium interactions: lithium and electroconvulsive therapy. *J Clin Psychopharmacol* 1990, 10:346–350.
11. Sackeim HA, Decina P, Malitz S, et al.: Postictal excitement following bilateral and right-unilateral ECT. *Am J Psychiatry* 1983, 140:1367–1368.
12. Swartz CM: ECT emergence agitation and methohexital-succinylcholine interaction. *Gen Hosp Psychiatry* 1993, 15:339–341.
13. Devanand DP, Sackeim HA: Use of increased anesthetic dose prior to electroconvulsive therapy to prevent postictal excitement. *Gen Hosp Psychiatry* 1992, 14:345–349.
14. Kelly K, Zisselman M: Update on electroconvulsive therapy (ECT) in older adults. *J Am Geriatr Soc* 2000, 48:560–566.
15. Levin L, Wambold D, Viguera A, et al.: Homodynamic responses to ECT in a patient with critical aortic stenosis. *J ECT* 2000, 16:52–61.
16. Zielinski R, Roose S, Devanand D, et al.: Cardiovascular complications of ECT in depressed patients with cardiac disease. *Am J Psychiatry* 1993, 150:904–909.
17. Lewis W, Richardson J, Gahagan L: Cardiovascular disturbances and their management in modified electrotherapy for psychiatric illness. *N Engl J Med* 1955, 252:1016–1020.
18. Dec W, Stern T, Welch C: The effects of electroconvulsive therapy on serial electrocardiograms and serum cardiac enzyme values. A prospective study of depressed hospitalized inpatients. *JAMA* 1985, 253:2525–2529.
19. Karliner W: Cardiovascular disease and ECT. *Psychosomatics* 1978, 19:238–241.
20. O'Reardon J, Cristancho M, Cristancho P, et al.: Electroconvulsive therapy in a 96 year old patient with severe aortic stenosis—a case report and review of the literature. *J ECT* 2008, 24:96–98.
- An interesting case report and review of literature that illustrates that neither age nor aortic stenosis by itself precludes ECT in the setting of severe depression.
21. O'Reardon J, Lott J, Akthar U, et al.: Acute coronary syndrome (Takotsubo cardiomyopathy) following electroconvulsive therapy in the absence of significant coronary artery disease: case report and review of literature. *J ECT* 2008 (in press).
- Interesting case report and review of literature presenting and discussing the first reported case of TCM in the setting of ECT administration.
22. Prasad A: Apical ballooning syndrome: an important differential diagnosis of acute myocardial infarction. *Circulation* 2007, 115:e56–e59.
23. Ueyama T, Kasamatsu K, Hano T, et al.: Emotional stress induces transient left ventricular hypocontraction in the rat via activation of cardiac adrenoceptors: a possible animal model of 'tako-tsubo' cardiomyopathy. *Circ J* 2002, 66:712–713.
24. Messina A, Parancas MKB, Markowitz J, et al.: Effect of electroconvulsive therapy on the electrocardiogram and echocardiogram. *Anesth Analg* 1992, 75:511–514.
25. Bybee KA, Kara T, Prasad A, et al.: Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. *Ann Intern Med* 2004, 141:858–865.
26. Tsuchihashi K, Ueshima K, Uchida T, et al.: Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. Angina Pectoris-Myocardial Infarction Investigations in Japan. *J Am Coll Cardiol* 2001, 38:11–18.
27. Sienaert P, Koeck A, Bouckaert F, et al.: Prolonged ECT seizure in a patient taking Nimesulide. *J ECT* 2004, 20:52–53.
28. Abrams R: *Electroconvulsive Therapy*, edn 4. New York: Oxford University Press; 2002.
29. American Psychiatric Association: *The Practice of ECT: Recommendations for Treatment, Training and Privileging*. Washington, DC: American Psychiatric Press; 2001.
30. Scott AI, McCreddie RG: Prolonged seizures detectable by electroencephalogram in electroconvulsive therapy. *Br J Psychiatry* 1999, 175:91–92.
31. Guttmacher LB, Cretella H: Electroconvulsive therapy in one child and three adolescents. *J Clin Psychiatry* 1988, 49:20–23.
32. Solomons K, Holliday S, Illing M: Non-convulsive status epilepticus complicating electroconvulsive therapy. *Int J Geriatr Psychiatry* 1998, 13:731–734.
33. Beyer JL, Glenn MD, Weiner RD: *Electroconvulsive Therapy: A Programmed Text*, edn 2. Washington, DC: American Psychiatric Press; 1998.
34. Proposal for revised clinical and electroencephalographic classification of epileptic seizures: from the Commission on Classification and Terminology of the International League Against Epilepsy. *Epilepsia* 1981, 22:489–501.
35. Mayer SA, Claassen J, Lokin J, et al.: Refractory status epilepticus frequency, risk factors, and impact on outcome. *Arch Neurol* 2002, 59:205–210.
36. Fink M, Kellner CH, Sackeim HA: Intractable seizures, status epilepticus, and ECT. *J ECT* 1999, 15:282–284.
37. Povlsen UJ, Wildschiodtz G, Hogenhaven H, et al.: Nonconvulsive status epilepticus after electroconvulsive therapy. *J ECT* 2003, 19:164–169.
38. Devinsky O, Duchowny MS: Seizures after convulsive therapy: a retrospective case survey. *Neurology* 1983, 33:921–925.
39. Balki M, Castro C, Ananthanarayan C: Status epilepticus after electroconvulsive therapy in a pregnant patient. *Int J Obstet Anesth* 2006, 15:325–328.
40. Lowenstein DH, Alldredge BK: Status epilepticus. *N Engl J Med* 1998, 338:970–976.
41. Cline JS, Roos K: Treatment of status epilepticus with electroconvulsive therapy. *J ECT* 2007, 23:30–32.
42. Treiman DM, Meyers PD, Walton NY, et al.: A comparison of four treatments for generalized convulsive status epilepticus. *N Engl J Med* 1998, 339:792–798.

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43. Scott AI, Riddle W: Status epilepticus after electroconvulsive therapy. *Br J Psychiatry* 1989, 155:119–121.
44. Smith K, Keepers G: Nonconvulsive status epilepticus after ECT. *Am J Psychiatry* 2000, 157:1524.
45. Pogarell O, Ehrentauf S, Rüther T, et al.: Prolonged confusional state following electroconvulsive therapy—diagnostic clues from serial electroencephalography. *Pharmacopsychiatry* 2005, 38:316–320.
46. Kaplan PW: Assessing the outcomes in patients with nonconvulsive status epilepticus: nonconvulsive status epilepticus is underdiagnosed, potentially overtreated, and confounded by comorbidity. *J Clin Neurophysiol* 1999, 16:341–352.
47. Fink M: Nonconvulsive status epilepticus and electroconvulsive therapy. *J ECT* 2004, 20:131–132.
48. Sackeim HA, Decina P, Prohovnik I, et al.: Anticonvulsant and antidepressant properties of electroconvulsive therapy: a proposed mechanism of action. *Biol Psychiatry* 1983, 18:1301–1310.
49. Griesemer DA, Kellner CH, Beale MD, et al.: Electroconvulsive therapy for treatment of intractable seizures. Initial findings in two children. *Neurology* 1997, 49:1389–1392.
50. Lisanby SH, Bazil CW, Resor SR, et al.: ECT in the treatment of status epilepticus. *J ECT* 2001, 17:210–215.

EXHIBIT 36



THE STATE EDUCATION DEPARTMENT / THE UNIVERSITY OF THE STATE OF NEW YORK / ALBANY, NY 12234

TO: EMSC-VESID Committee

FROM: Rebecca H. Cort *Rebecca H. Cort*

SUBJECT: Policy on the Use of Aversive or Noxious Stimuli in Public and Private Schools Serving Students with Disabilities

DATE: May 16, 2006

STRATEGIC GOAL: Goals 1 and 2

AUTHORIZATION(S): *R. Cort*

Executive Summary

Issue for Discussion

Should the Regents adopt emergency regulations to prohibit or limit the use of certain behavioral approaches, including the use of certain aversive or noxious stimuli to reduce or eliminate maladaptive behaviors of students?

Proposed Handling

Discussion

Procedural History

This issue was first discussed at the March 2006 EMSC/VESID Committee Meeting.

Background Information

Currently, neither Education Law nor Commissioner's Regulations prohibit the use of aversive behavioral interventions in school programs, although the Rules of the Board of Regents prohibit corporal punishment. Several recent events and information have heightened the Department's concerns on this issue and have led us to bring this issue to the attention of the Board of Regents at this time. These events include changes to federal law, the enactment of Billy's Law, and a proposed Senate bill that would prohibit the use of aversives in certain school programs.

There is currently a lack of a clear policy and no standards or guidance on this issue at a time when we are assessing the extent of the use of aversive interventions at public and private school programs. We believe that greater guidance and oversight is necessary to ensure the availability of professional advice to districts and parents when making the difficult decision as to whether all possible alternatives to aversive interventions have been considered and, where aversive interventions are determined to be absolutely necessary, on how to minimize their intensity and duration.

Recommendation

It is recommended that the Board of Regents discuss and provide direction regarding emergency regulations to establish:

1. general rules for behavioral interventions, including a prohibition on the use of aversive or noxious stimuli as a consequence for behaviors;
2. a process for exceptions to the prohibition on the use of aversive behavioral interventions on a child-specific basis; and
3. standards for programs using aversive interventions, as authorized through the child-specific waiver process.

These regulations would require that school districts seek the advice of a panel of experts, comprised of a minimum of three professionals with appropriate clinical and behavioral expertise, prior to considering whether a child-specific exception to use aversive behavioral interventions should be permitted; establish standards for the use of aversive behavioral interventions when provided through a child-specific exception; and require the Department to be fully informed when any public or private school program includes aversive interventions as a means of addressing student behaviors.

Timetable for Implementation

The Board will discuss the regulations in May and be asked to act on proposed emergency regulations at the June Regents meeting.

Attachment

The Use of Aversive or Noxious Stimuli in Public and Private Schools Serving Students with Disabilities

At the March 2006 Regents meeting, the Board reviewed issues to consider in discussing and developing a policy on the use of aversive and noxious stimuli. These included the following:

- All New York State (NYS) children deserve to be treated with respect and dignity in the course of their receipt of appropriate education programs and services.
- A student's right to a free appropriate public education must be ensured. States and local educational agencies must ensure a full continuum of services and have programs available to meet the needs of individual students with varying and complex needs.
- The use of aversive therapy is a very controversial issue that is polarizing parents and professionals.
- It is important that we are knowledgeable about the most current research prior to establishing policy on the efficacy and research-based standards for the use of aversive interventions as a behavior management approach to effectively reduce or eliminate maladaptive behaviors on more than a temporary basis.
- There is general concern among NYS agencies with responsibility for the care and treatment of children surrounding the use of aversive approaches. As Regents policy is developed, we must consider any potential conflict with the policies and procedures relating to aversive treatments of other NYS agencies that operate educational programs or license the residential components of State Education Department (SED) approved schools (e.g., Office of Mental Health (OMH), Office of Mental Retardation and Developmental Disabilities (OMRDD)).
- Any policy regarding the use of aversive therapies must be uniformly applied to all schools where NYS students attend, including public schools, boards of cooperative educational services (BOCES), approved preschool programs, approved private schools, including day and residential schools, Charter Schools, and State-operated and State-supported schools.

What are the compelling factors for a Board of Regents policy on the use of behavioral interventions?

- The Individuals with Disabilities Education Act (IDEA) requires the individualized education programs (IEPs) of students with disabilities to include positive behavioral supports and services and functional behavioral assessments and behavioral intervention programs for students with behaviors which impede learning.
- The use of aversive behavioral approaches is the most extreme and intrusive intervention that could be used in educational programs. The unregulated use of

such interventions has potential for compromising the health and safety and the physical and psychological well being of students.

- Absent regulatory parameters, the current practice on the use of aversive behavioral interventions affecting NYS students includes a variety of methodologies that are unrestricted and are based on individual agency or practitioner practices and not necessarily based on, nor implemented consistent with, acceptable research-based standards. Currently, aversive interventions are used in a wide range of varying circumstances.
- SED establishes requirements for the educational environment conducive to learning, appropriate provision of services including such areas as instructional class sizes based on the intensity of the students' needs, and grouping based on educational need. It is inconsistent that we do not regulate behavioral interventions, which have the potential to significantly impact the health and safety of our students.
- Regulations in this area will strengthen SED's authority to ensure appropriate provision of services consistent with peer-reviewed research practices and to protect the health and safety of NYS students consistent with our oversight and supervision responsibilities.
- The Board of Regents should identify its direction on this issue to ensure that any standards established in State law or through activities of the out-of-state Placement Committee are developed in consideration and are consistent with the policy direction of the Board of Regents.

Other Findings to Consider

Prevalence in the use of aversive or noxious stimuli in public and in-state and out-of-state approved private schools serving NYS students with disabilities

- The Office of Vocational and Educational Services for Individuals with Disabilities (VESID) surveyed all public and approved private schools, both in-state and out-of-state, serving NYS students requesting a response as to whether the school uses or has a policy that allows for the use any of the aversive or noxious stimuli as such term was described in the March 2006 Regents report. To date, all NYS public schools that have responded have indicated that they do not directly use such interventions. However, public school districts contract for aversive interventions when they place students in certain approved private school programs, including certain preschool programs.
- We are aware of at least three approved private school programs that do use some forms of aversive interventions, two of which are preschool programs and one is an out-of-state residential school. There may be additional schools that use such interventions, as we have not yet heard from all the private schools, including all the in-state preschool programs and a number of out of state residential programs.

- The identified preschool programs are known to use over correction, noxious sprays, odors and/or tastes as consequences for inappropriate behaviors.
- The policy of the residential school identifies aversive interventions used as consequences for inappropriate behaviors to include but not be limited to:
 - *Manual and mechanical movement limitation* (e.g., "leg, waist or crossover restraints, an arm-free or four-point chair, four-point restraint boards, arm splints, arm tubes, helmet or visual screen goggles; specially designed helmets with Plexiglas or grid-type face guards and/or mechanisms which prevent removal).
 - *Contingent food programs* based on a diet of "preferred staple foods" at a targeted number of calories per day with non-preferred staple food (bland food consisting of mashed potatoes, chicken and spinach garnished with liver powder) used as a consequence of negative behaviors.
 - *Electrical stimulation* through a Graduated Electronic Decelerator or "GED" which is manufactured by the Judge Rotenberg Educational Center (JRC). The GED has adjustable intensities and remote distanced electrodes (two electrodes mounted up to six inches apart to "increase the GED's therapeutic value").
- The recommendations to use aversive interventions were primarily initiated at the request of agencies serving the students in the first instance and were not the determinate factor for a Committee on Special Education (CSE) or Committee on Preschool Special Education (CPSE) to pursue a particular private school program for a student. In some cases, the IEPs of students did not include CSE or CPSE recommendations for aversive interventions.

Experiences of other states with laws and regulations addressing the use of aversive or noxious stimuli in school programs

- We have identified eleven states that have statutory and/or regulatory limitations on behavioral interventions, and six that specifically prohibit the use of aversive interventions. States with prohibitions on the use of aversive or noxious interventions have established a specific definition of the term "aversive" either in law or regulations. Most state prohibitions on the use of aversive interventions provide for a process for individual exceptions under certain circumstances.
- California has the longest history of such a statute ("Hughes Act") and regulations, adopted in the early 1990s as a result of the death of a child stemming from a form of restraint. California's Education statute and regulations prohibit the use of procedures that cause pain or trauma to eliminate maladaptive behaviors. California law includes a child-specific waiver provision through which exceptions to state statute can be approved on a child-specific basis by a Waiver Board appointed by the Governor. Since the law went into effect, the Waiver Board reviewed only two requests for child specific waivers on the use of aversive interventions.

- We have identified three states, including New York, with pending legislation that would prohibit aversive interventions. At the federal level, there is a bill in discussion draft form that would amend the Public Health Service Act to require annual reports from residential treatment facilities that receive funds under the Medicaid program with respect to the use of restraint and seclusion procedures and the use of electric shock.

Policies of other NYS agencies

- Representatives from the State agencies represented on the NYS Out-of-State Placement Committee established by Chapter 392 of the Laws of 2005 ("Billy's Law") are discussing a standard policy on the use of behavioral interventions for NYS children placed in congregate care facilities.
- The potential conflict with the policies and procedures relating to aversive treatments of other NYS agencies that operate educational programs or license the residential components of SED approved schools (e.g., the Office of Children and Family Services, OMH and OMRDD) must be considered in the development of Regents policy. OMRDD, for example, has draft regulations on the use of aversive interventions in limited circumstances that would require several layers of clinical review, including a review by the Commissioner, prior to such use. Programs operated by other NYS agencies (e.g., education programs in State-operated psychiatric centers or developmental centers) should be governed by the rules of the State agencies operating those education programs.

Building in-state capacity to serve NYS students with disabilities

SED has taken significant steps to increase the capacity, availability and appropriateness of in-state programs to serve NYS students with disabilities.

- Eleven agencies have submitted letters of intent to develop in-state residential programs for high need students with disabilities.
- A new Residential Capacity Notification System has been implemented so that school districts can readily access in-state vacancy information prior to referring students to programs in other states.

VESID is exploring a new model of short-term educational placements where students can receive intensive research-based behavioral and educational interventions designed and implemented to modify behaviors so that children can benefit from other day or residential programs. We are researching the effectiveness and possible replications of such programs as used in other states (e.g., the Kennedy Krieger Institute in Maryland).

Unsolicited letters and public comment

SED, individual members of the Board of Regents and Legislators have received numerous unsolicited letters and telephone calls relating to the issue of the use of

aversive interventions. Some of the letters are from parents of students or adults attending JRC supporting continued approval of this school for NYS students. Other letters have been from professionals, the majority of which have expressed concern and opposition for the use of aversive interventions. Copies of these letters are available for review by the Regents upon request to the Secretary to the Board of Regents.

Positive Behavioral Interventions

- Most of the research in the past decade has focused on positive behavioral interventions and it is well documented in the scientific literature that positive behavioral interventions have been effective in addressing behaviors, including self-abusive behaviors. Long-term studies show that when positive interventions are employed, the rate of maintaining positive behaviors is sustained far more effectively than in cases where negative consequences or punishment are used.
- Many of the older individuals from NYS currently served by JRC and for whom aversive interventions are currently being used did not have the opportunity to benefit from programs using peer-reviewed research on positive behavioral interventions that has emerged over the past decade.
- We were unable to identify any peer-reviewed research which supports the interventions as used at JRC, including the use of GED on the population of students and at the intensities, frequency, circumstances, types of behaviors and duration (in some cases for more than three years) for which they are used at JRC.

Recommendations

It is recommended that the Board of Regents discuss and provide direction regarding emergency regulations to establish:

1. general rules for behavioral interventions, including a prohibition on the use of aversive or noxious stimuli as a consequence for behaviors;
2. a process for exceptions to the prohibition on the use of aversive behavioral interventions on a child-specific basis; and
3. standards for programs using aversive interventions, as authorized through the child-specific waiver process.

The proposed regulations would be developed in consultation with other State agencies serving children and including representatives of agencies represented on the Out-of-State Placement Committee.

General rules for behavioral interventions

- Prohibit a teacher, administrator, officer, employee or agent of a school district in this State, or of a BOCES, Charter School, approved preschool program, approved private school, State-operated or State-supported school in this State or an

approved out-of-state day or residential school, from using aversive or noxious stimuli to reduce or eliminate maladaptive behaviors. Provide an exception for education programs operated pursuant to section 112 of the Education Law (e.g., OMH- and OMRDD-operated education programs).

- Establish guidelines on the use of positive behavioral interventions, including regulations related to the development and use of functional behavioral assessments, the development, implementation, modification and monitoring of behavioral intervention plans, the role of the CSE/CPSE and required documentation on the IEP.
- Define emergency interventions, including the use of restraints, and establish rules on the use of restraints, including staff development, monitoring and reporting.

Exceptions on a child-specific basis

- In the event a CSE or CPSE determines that a student may require the use of aversive behavioral interventions in order to provide a student with a free appropriate public education:
 - Provide a process by which a school district could submit to SED a request for a time-limited child-specific exception on the prohibition of the use of aversive behavioral interventions. This exception process would ensure that the prohibition on the use of such interventions does not abrogate any right of the student provided in IDEA.
 - Establish the conditions upon which such waivers could be granted by the Commissioner based on a recommendation of a review board of experts.
 - Ensure that the school district submit periodic reports of the student's behavioral intervention program as authorized through the waiver process, which must include reports of the student's progress and the recommendations and results to fade the use of such interventions.
 - Ensure that aversive interventions are provided only with the informed written consent of the parent; that parents may withdraw their consent for the use of such interventions at any time; and that other procedures established to authorize the use of such interventions do not abrogate the rights of the parent or guardian regarding authorization and consent.
 - Ensure that representatives of the school district periodically observe and regularly review the application of such interventions, consistent with recommendations documented on the student's IEP and behavioral intervention plan approved by the CSE or CPSE.

Standards for programs using aversive interventions

- Establish standards for programs using aversive interventions as allowed through the child-specific waiver process, including but not limited to regulations that would ensure:
 - Aversive behavioral intervention procedures shall not be used in the absence of other therapies, including verbal or other counseling therapies and functional communication training.
 - Regular and frequent monitoring and reporting to the CSE or CPSE and to the child-specific waiver review panel.
 - Aversive interventions are monitored and administered only by appropriately qualified personnel.
 - Aversive interventions are administered in a manner that protects the privacy, human dignity and rights of students.
- Require that a behavioral intervention plan that includes aversive interventions be implemented consistent with peer-reviewed research based practices.
- Require any school that uses aversive interventions to submit its behavioral intervention policies and procedures, and any modifications of such policies and procedures, for prior approval by SED to ensure such policies are consistent with standards and peer reviewed research-based practices.

Summary of Draft Regulations on Behavioral Interventions

The attached draft proposed regulations would establish:

1. general rules for behavioral interventions, including a prohibition on the use of aversive or noxious stimuli as a consequence for behaviors;
2. a process for exceptions to the prohibition on the use of aversive behavioral interventions on a child-specific basis; and
3. standards for programs using aversive interventions, as authorized through the child-specific waiver process.

Following is a summary of these draft proposed regulations.

Section 19.5 of Chapter 1 of the Rules of the Board of Regents would be amended to:

- extend the prohibition on the use of corporal punishment to registered nonpublic nursery, kindergarten, elementary or secondary schools.
- add a prohibition on the use of aversive behavioral interventions, except as provided through a child-specific exception process established in a new proposed section 200.22 of the Commissioner's Regulations.
- define the term "aversive behavioral interventions."

Section 200.1 of the Commissioner's Regulations would be amended to:

- add a definition of the term "aversive behavioral interventions."
- add a definition of the term "behavioral intervention plan."

Section 200.4 of the Commissioner's Regulations would be amended to:

- add a requirement that the committee on special education (CSE) or committee on preschool special education (CPSE) consider behavioral interventions that are consistent with the new proposed regulations on program standards for behavioral interventions.

Section 200.7 of the Commissioner's Regulations relating to approval and standards for private schools and State-operated and State-supported schools would be amended to:

- require approval of the school's policies and procedures for behavioral interventions when a private school applying for approval proposes to use aversive behavioral interventions.
- indicate that a school may be removed from the approved list for providing aversive behavioral interventions without a child-specific exception or in a manner inconsistent with the Department's programs standards for such use.
- prohibit the use of aversive behavioral interventions for students without a child-specific exception.
- for currently approved schools that use or propose to use aversive behavioral interventions, require submission of the school's policies and procedures for aversive behavioral interventions to the Department by August 15, 2006 and establish consequences for the failure to comply with this requirement.

A new section 22 would be added to Part 200 of the Commissioner's Regulations regarding program standards for behavioral interventions relating to:

- the development of functional behavioral assessments.
- the development of behavioral intervention plans.
- standards for the use of time out rooms.
- standards for the emergency use of physical restraints.
- a process for providing child-specific exceptions to use aversive behavioral interventions to reduce or modify student behaviors, which would include review by an independent panel of professionals with appropriate clinical and behavioral expertise to provide a recommendation to the CSE or CPSE if an exception is necessary so as not to hinder the student's right to a free, appropriate public education.
- program standards for the use of aversive behavioral interventions that include requirements for:
 - the humane and dignified treatment of students;
 - procedures used consistent with a student's individualized education program (IEP) and behavioral intervention plan;
 - the use of aversive behavioral intervention procedures in conjunction with other therapies;
 - the use of reinforcement procedures based on student reinforcement preferences;
 - the application of aversive behavioral interventions consistent with peer-reviewed research that include procedures for a student to generalize and maintain behaviors and for the fading of aversive behavioral interventions;
 - limiting the use of aversive behavioral interventions to those behaviors of greatest concern;
 - use of the aversive interventions at the lowest intensity and for the shortest duration and period of time effective to treat the problem behavior and use of strategies that increase the effectiveness of mild levels of aversive interventions;
 - approval of the US Food and Drug Administration for any aversive conditioning devices and a requirement that the magnitude, frequency and duration for the use of such a device must have been shown to be safe and effective in clinical peer-reviewed studies.
 - establishment of a Human Rights Committee in each program that uses aversive behavioral interventions;
 - appropriate certification, licensing, supervision and training of all personnel who will be authorized to use aversive behavioral interventions;
 - informed parent consent prior to the use of aversive behavioral interventions;
 - progress monitoring of aversive behavioral intervention plans, including quarterly reports submitted to the CSE or CPSE; and
 - school district oversight responsibilities for students receiving aversive behavioral interventions, including a requirement for a CSE or CPSE meeting at least every six months.
- An exception for education programs operated by another State agency where the rules of the other State agencies are inconsistent with the requirements of this section.

draft

AMENDMENT TO THE REGULATIONS OF THE COMMISSIONER OF EDUCATION

Pursuant to Education Law sections 207, 210, 4401, 4402, 4403, and 4410

1. Section 19.5 of the Chapter I of the Rules of the Board of Regents is amended, effective _____, 2006, as follows:

§ 19.5 Prohibition of corporal punishment and certain behavioral interventions.(a) Prohibition of corporal punishment

(1) No teacher, administrator, officer, employee or agent of a school district in this State, [or of] a board of cooperative educational services (BOCES), a charter school, an approved preschool program, an approved private school, an approved out-of-State day or residential school, or of a registered nonpublic nursery, kindergarten, elementary or secondary school in this State, shall use corporal punishment against a pupil.

[(b)] (2) As used in this section, corporal punishment means any act of physical force upon a pupil for the purpose of punishing that pupil, except as otherwise provided in [subdivision (c)] paragraph 3 of this [section] subdivision.

[(c)] (3) In situations in which alternative procedures and methods not involving the use of physical force cannot reasonably be employed, nothing contained in this section shall be construed to prohibit the use of reasonable physical force for the following purposes:

[(1)] (i) to protect oneself from physical injury;

[(2)] (ii) to protect another pupil or teacher or any person from physical injury;

[(3)] (iii) to protect the property of the school, school district or others; or

[(4)] (iv) to restrain or remove a pupil whose behavior is interfering with the orderly exercise and performance of school or school district functions, powers and duties, if that pupil has refused to comply with a request to refrain from further disruptive acts.

(b) Prohibition of the use of aversive behavioral interventions.

(1) No public school, BOCES, charter school, approved preschool program, approved private school, State-operated or State-supported school in this State, approved out-of-State day or

residential school, or registered nonpublic nursery, kindergarten, elementary or secondary school in this State shall employ the use of aversive behavioral interventions to reduce or eliminate maladaptive behaviors, except as provided pursuant to section 200.22(e) and (f) of this Title.

(2) As used in this section, aversive behavioral intervention means:

- (i) application of noxious, painful, intrusive stimuli or activities intended to induce pain such as electric skin shock, ice applications, hitting, slapping, pinching, kicking, hurling, strangling, shoving, deep muscle squeezes or other similar stimuli;
- (ii) any form of noxious, painful or intrusive spray or inhalant;
- (iii) withholding sleep, shelter, bedding, bathroom facilities or clothing;
- (iv) contingent food programs that include withholding or limiting food or drink or essential nutrition or hydration as part of meal times or intentionally altering staple food or drink in order to make it distasteful;
- (v) movement limitation used as a punishment, including but not limited to helmets and mechanical restraint devices;
- (vi) the placement of a child unsupervised or unobserved in a room from which the student cannot exit without assistance;
or
- (vii) other stimuli or actions similar to the interventions described in subparagraphs (i) through (vi) of this paragraph.

The term does not include such interventions as voice control, limited to loud, firm commands; time-limited ignoring of a specific behavior; token fines as part of a token economy system; brief physical prompts to interrupt or prevent a specific behavior; or other similar interventions.

2. Paragraphs (lll) and (mmm) are added to section 200.1 of the Regulations of the Commissioner of Education, effective _____, as follows:

(III) Aversive behavioral intervention means application of noxious, painful, intrusive stimuli or activities intended to induce pain such as electric skin shock, ice applications, hitting, slapping, pinching, kicking, hurling, strangling, shoving, deep muscle squeezes or other similar stimuli; any form of noxious, painful or intrusive spray or inhalant; withholding sleep, shelter, bedding, bathroom facilities or clothing; contingent food programs that include withholding or limiting food or drink or essential nutrition or hydration as part of meal times or intentionally altering staple food or drink in order to make it distasteful; movement limitation used as a punishment, including but not limited to helmets and mechanical restraint devices; the placement of a child unsupervised or unobserved in a room from which the student cannot exit without assistance; or other similar stimuli or actions. The term does not include such interventions as voice control, limited to loud, firm commands; time-limited ignoring of a specific behavior; token fines as part of a token economy system; brief physical prompts to interrupt or prevent a specific behavior; or other similar interventions.

(mmm) Behavioral intervention plan means a plan that is based on the results of the functional behavioral assessment and, at a minimum, includes a description of the problem behavior, global and specific hypotheses as to why the problem behavior occurs and intervention strategies to address the behavior.

3. Subparagraph (i) of paragraph (3) of subdivision (d) of section 200.4 of the Regulations of the Commissioner of Education is amended, effective _____, as follows:

- (i) in the case of a student whose behavior impedes his or her learning or that of others, consider strategies, including positive behavioral interventions, and supports and other strategies to address that behavior that are consistent with the requirements in section 200.22 of this Part;

4. Subparagraph (i) of paragraph (2) of subdivision (a) of section 200.7 of the Regulations of the Commissioner is amended, effective _____, 2006, as follows:

- (i) Conditional approval for private schools shall be limited to a period of one school year, or the period of time required to complete approval, and will be based on:

- (a) . . .

- (b) . . .
 - (c) . . .
 - (d) For schools operating as corporate entities, evidence of the following:
 - (1) . . .
 - (2) . . .
 - (3) for out-of-state schools, a license or charter from the state education agency of the state in which the school is located; [and]
 - (e) at least one onsite program review visit by program or fiscal staff of the Education Department; and
 - (f) submission for approval of the school's procedures regarding behavioral interventions, including, if applicable, procedures for the use of aversive behavioral interventions.
5. Subparagraph (iv) of paragraph (3) of subdivision (a) of section 200.7 of the Regulations of the Commissioner of Education is amended, effective _____, 2006, as follows:
- (iv) Schools may be removed from the approved list five business days after written notice by the commissioner indicating that there is a clear and present danger to the health or safety of students attending the school, and listing the dangerous conditions at the school, including, but not limited to, evidence that an approved private school is using aversive behavioral interventions to reduce or eliminate maladaptive behaviors of students without a child-specific exception provided pursuant to section 200.22(e) of this Part or that an approved private school is using aversive behavioral interventions in a manner inconsistent with the standards as established in section 200.22 (f) of this Part.
6. Paragraph (8) is added to subdivision (b) of section 200.7 of the Regulations of the Commissioner of Education, effective _____ 2006, as follows:
- (8) Except as provided in subdivision (e) of section 200.22 of this Part, an approved private school, a State-operated school, or a State-supported school is prohibited from using corporal punishment and aversive behavioral interventions to reduce or eliminate maladaptive behaviors of students.

7. Paragraph (6) is added to subdivision (c) of section 200.7 of the Regulations of the Commissioner of Education, effective _____, 2006, as follows:

(6) Policies and procedures relating to the use of aversive behavioral interventions. Not later than August 15, 2006, a private school that proposes to use or continue to use aversive behavioral interventions in its program shall submit its written policies and procedures on behavioral interventions to the Department with certification that the school's policies, procedures and practices are demonstrably in compliance with the standards established in section 200.22(f) of this Part. Any school that fails to meet this requirement shall be immediately closed to new admissions of New York students and shall be prohibited from using aversive behavioral interventions with any New York State student placed in such program. Failure to comply with this requirement may result in termination of private school approval pursuant to paragraph (3) of subdivision (a) of this section.

8. A new section 200.22 is added to Part 200 of the Regulations of the Commissioner of Education, effective _____, 2006, as follows:

§ 200.22 Program standards for behavioral interventions.

Behavioral intervention plans shall be provided in accordance with this section and those other applicable provisions of this Part that are not inconsistent with this section.

(a) Assessment of student behaviors. For purposes of this section, an assessment of student behaviors shall mean a functional behavioral assessment (FBA), as such term is defined in section 200.1(r) of this Part.

(1) A FBA shall be conducted as required in section 200.4 of this Part and section 3 of Part 201 of this Title.

(2) The FBA shall, as appropriate, be based on information obtained from direct observation of the student, information from the student, the student's teacher(s) and/or related service provider(s), a review of available data and information from the student's record and other sources including any relevant information provided by the student's parent. The

FBA shall not be based solely on the student's history of presenting problem behaviors.

- (3) The FBA shall provide a baseline of the student's problem behaviors across activities, settings, people and times of the day and include the information required in section 200.1(r) of this Part in sufficient detail to form the basis for a behavioral intervention plan for the student that addresses antecedent behaviors, reinforcing consequences of the behavior, recommendations for teaching alternative skills or behaviors and an assessment of student preferences for reinforcement.

(b) Behavioral intervention plan. The CSE or CPSE shall consider the development of a behavioral intervention plan for a student with a disability whenever the student's behaviors that impede the student's learning or that of others persist despite consistently implemented general school-wide or classroom-wide interventions; when the student's behavior places the student or others at risk of harm or injury; when the CSE or CPSE is considering more restrictive programs or placements as a result of the student's behavior; and as required pursuant to section 201.3 of this Title.

- (1) In accordance with the requirements in section 200.4 of this Part, in the case of a student whose behavior impedes his or her learning or that of others, the CSE or CPSE shall consider strategies, including positive behavioral interventions and supports and other strategies to address that behavior. The IEP shall include a statement if the student needs a particular device or service, including an intervention, accommodation or other program modification in consideration of the student's behavior that impedes his or her learning or that of others. A student's need for a behavioral intervention plan shall be documented on the IEP and such plan shall be reviewed at least annually by the CSE or CPSE.
- (2) Except as provided in subdivision (f) of this section, a behavioral intervention plan shall not include the use of aversive behavioral interventions.
- (3) The behavioral intervention plan shall identify:

- (i) the baseline measure of the problem behavior, including the frequency, duration and intensity of the targeted behaviors. Such baseline shall, to the extent practicable, include data taken across activities, settings, people and times of the day. The baseline data shall be used as a standard against which to evaluate intervention effectiveness;
 - (ii) the intervention strategies to be used to alter antecedent events to prevent the occurrence of the behavior, teach individual alternative and adaptive behaviors to the student, and provide consequences for the targeted inappropriate behavior(s) and alternative acceptable behavior(s); and
 - (iii) a schedule to measure the effectiveness of the interventions, including the frequency, duration and intensity of the targeted behaviors at scheduled intervals.
 - (4) Progress Monitoring. The implementation of a student's behavioral intervention plan shall include regular progress monitoring of the frequency, duration and intensity of the behavioral interventions at scheduled intervals, as specified in the behavioral intervention plan and on the student's IEP. The results of the progress monitoring shall be documented and reported to the student's parents and to the CSE or CPSE and shall be considered in any determination to revise a student's behavioral intervention plan or IEP.
- (c) Use of time out rooms. (1) Each school which uses a time out room as part of its behavior management approach shall ensure that the school's policy and procedures on the use of the time out room are developed and implemented consistent with State policy, including the physical and monitoring requirements, parental rights and IEP requirements for students with disabilities.
- (2) A student's IEP shall specify when a behavioral intervention plan includes the use of a time out room for a student with a disability, including the maximum amount of time a student will need to be in a time out room as a behavioral consequence as determined on an individual basis in consideration of the student's age and individual needs.

- (3) Except for emergency interventions, the use of a time out room shall only be used in conjunction with a behavioral intervention plan that is designed to teach and reinforce alternative appropriate behaviors.
- (4) Parents shall be informed prior to the initiation of a behavioral intervention plan which will incorporate the use of a time out room. Upon request, parents must be shown the physical space that will be used as a time out room.
- (5) The physical space used as a time out room shall provide a means for continuous visual and auditory monitoring of the student. The room shall be of adequate width, length and height to allow the student to move about and recline comfortably. Wall and floor coverings should be designed to prevent injury to the student and there shall be adequate lighting and ventilation. The temperature of the room shall be within the normal comfort range and consistent with the rest of the building. The room shall be clean and free of objects and fixtures that could be potentially dangerous to a student and shall meet all local fire and safety codes.
- (6) The time out room shall be unlocked and the door must be able to be opened from the inside. The use of locked rooms or spaces for purposes of time out is prohibited.
- (7) Staff shall be assigned to continuously monitor the student in a time out room. The staff must be able to see and hear the student at all times.
- (8) The school shall establish and implement procedures to document the use of the time out room, including information to monitor the effectiveness of the use of the time out room to decrease specified behaviors.
- (9) The use of time out rooms in education programs operated pursuant to section 112 of the Education Law and Part 116 of the Regulations of the Commissioner of Education are subject to the rules of the respective State agency operating such programs where such rules are inconsistent with the requirements in this section.

(d) Emergency use of physical restraints.

(1) The use of physical force to restrain a student from engaging in behaviors shall not be used as a substitute for systematic behavioral interventions that are designed to change, replace, modify or eliminate a targeted behavior.

(2) Staff who may be called upon to implement emergency interventions shall be provided with appropriate training in safe and effective restraint interventions.

(3) Emergency use of physical restraints shall only be used when other methods attempted to prevent escalation of the student's behaviors are ineffective to control the situation.

(4) Emergency interventions shall not include locked seclusion.

(5) The use of emergency interventions in education programs operated pursuant to section 112 of the Education Law and Part 116 of the Regulations of the Commissioner of Education are subject to the rules of the respective State agency operating such programs where such rules are inconsistent with the requirements in this section.

(e) Child-specific exception to use aversive behavioral interventions to reduce or modify student behaviors. Whenever a school district is considering the need for a child-specific exception to use aversive behavioral interventions as otherwise prohibited by section 19.5(b) of the Rules of the Board of Regents, the school district shall request a State-level review of such recommendation.

(1) Effective on or after October 1, 2006, when a CSE or CPSE is first considering a recommendation to use aversive behavioral interventions with a student, the school district shall submit an application for a recommendation for a child-specific exception to the prohibition of the use of aversive behavioral interventions, provided however that for any student with an IEP in effect prior to October 1, 2006 that includes a recommendation for an aversive behavioral intervention, such application shall be submitted prior to the next scheduled review of the student's IEP but not later than October 1, 2006 and annually thereafter.

- (2) The application for the child-specific exception, which shall be on a form prescribed by the commissioner, shall be reviewed by an independent panel of experts appointed by the commissioner or designee.
- (3) The review panel shall be comprised of a minimum of three professionals with appropriate clinical and behavioral expertise to make such determinations.
- (4) The child-specific review panel shall conduct a review of the written application, which shall include a review of the student's IEP, functional behavioral assessment, proposed, current and prior behavioral intervention plans, including documentation of the implementation and progress monitoring of the effectiveness of such plans, and other relevant individual evaluations and medical information, including any information provided by the student's parent.
- (5) The decision of the child-specific review panel to recommend that the CSE or CPSE provide an exception shall be based on the professional judgment of the review panel as to whether:

 - (i) the student is displaying self-abusive or aggressive behaviors that threaten the physical well being of the student or that of others; and
 - (ii) positive behavioral interventions have been consistently employed over an appropriate period of time and have failed to result in sufficient improvement of a student's behavior; or
 - (iii) a determination that such behaviors pose such significant health and safety concerns as to warrant the use of aversive behavioral interventions to affect change in the behavior.
- (6) The child-specific review panel shall, within 15 business days of receipt of an application, notify the school district and the commissioner of its recommendation.
- (7) The child-specific review panel may recommend, in whole or in part, that a child-specific exception be provided to use aversive behavioral interventions to reduce or modify a student's behaviors when the facts indicate that failure to do so would hinder the student's right to a free, appropriate public education.

- (i) A recommendation that supports a child-specific exception for the use of aversive behavioral interventions shall be based on the unanimous recommendation of the panel and shall identify the reasons for the recommendation.
- (ii) A recommendation to deny a child-specific exception shall identify the reasons why the child-specific exception should not be allowed by the CSE/CPSE.
- (8) The CSE or CPSE shall meet to review the recommendation of the child-specific review panel and make a recommendation concerning the use of aversive behavioral interventions for a student with a disability.
- (9) Any recommendation on a student's IEP to use aversive behavioral interventions shall identify the specific targeted behavior(s) and the specific aversive behavioral intervention to be used to address the behavior(s) and, specify if the aversive behavioral intervention includes the use of an aversive conditioning device.
- (10) Any recommendation by the child-specific review panel for an exception to the prohibition on the use of aversive behavioral interventions shall expire on the last day of the time period specified in the recommendation which shall not be more than one year. To extend the period of the exception, the CSE or CPSE must resubmit an application for review by the child-specific review panel of the progress of the student, which shall include the progress monitoring data of the implementation of the student's behavioral intervention plan.
- (11) Nothing in this section shall authorize the use of aversive behavioral interventions without the informed written consent of the student's parent.
- (12) A CSE or CPSE may recommend on a student's IEP to discontinue the use of aversive behavioral interventions with a student without informing the child-specific review panel.
- (13) The use of aversive behavioral interventions in education programs operated pursuant to section 112 of the Education Law and Part 116 of this Title are subject to the rules of the respective State agency operating

such programs where such rules are inconsistent with the requirements in this section.

- (14) Coordination with licensing agencies. Nothing in this section shall authorize a school or agency to provide aversive behavioral interventions that are otherwise prohibited by the State agency licensing such program.
- (f) Program standards for the use of aversive behavioral interventions.
- (1) Applicability. The requirements in this subdivision shall apply to a public school, BOCES, charter school, approved preschool program, approved private school, State-operated or State-supported school in this State and an approved out-of-State day or residential school.
- (2) In general. Any program that employs the use of aversive behavioral interventions to modify an individual student's behavior as authorized pursuant to subdivision (e) of this section shall comply with the following standards. The program's policies and procedures consistent with these standards shall be submitted to the Department for approval prior to the use of such interventions.
- (3) The program shall provide for the humane and dignified treatment of the student and for the development of such student's full potential at all times. The program shall promote respect for the student's personal dignity and right to privacy and shall not employ the use of threat of force, ridicule or humiliation, nor implement behavioral interventions in a manner that shows a lack of respect for basic human needs and rights.
- (4) Aversive behavioral intervention procedures may only be used if such interventions are recommended by the CSE or CPSE consistent with the student's IEP and behavioral intervention plan as determined by the CSE or CPSE.
- (5) Aversive behavioral intervention procedures shall not be used in the absence of other therapies such as verbal or other counseling therapies, speech and language therapy and/or functional communication training.
- (6) Aversive behavioral interventions shall be combined with reinforcement procedures, as individually determined based on an assessment of the student's reinforcement preferences.

- (7) Aversive behavioral interventions shall be implemented consistent with peer-reviewed research based practices and shall include individualized procedures for generalization and maintenance of behaviors and for the fading of the use of such aversive behavioral interventions.
- (8) The use of aversive behavioral interventions shall be limited to those behaviors of greatest concern as identified on the student's IEP.
- (9) Whenever possible, the use of aversive behavioral interventions shall apply the lowest intensity for the shortest duration and period of time that is effective to treat the problem behavior and employ strategies that increase the effectiveness of mild levels of aversive behavioral interventions. In the event the aversive behavioral intervention fails to result in a suppression of a behavior over time, alternative procedures shall be considered that do not include increasing the magnitude of the aversive behavioral intervention.
- (10) The use of any aversive conditioning device used to administer an electrical shock or other noxious stimuli to a student to modify undesirable behavioral characteristics shall be limited to devices tested for safety and efficacy and approved for such use by the United States Food and Drug Administration where such approval is required by federal regulation. The magnitude, frequency and duration of any administration of aversive stimulus from such a device must have been shown to be safe and effective in clinical peer-reviewed studies.
- (11) The program shall provide for ongoing monitoring of student progress, including the collection and review of data and information. Such information shall include reports on the indirect or collateral effects the use of aversive behavioral interventions may be having, including, but not limited to, increases in aggressive or escape behaviors, health-related effects and/or emotional reactions.
- (12) Human Rights Committee. (i) Each school that uses aversive behavioral interventions with students shall establish a Human Rights Committee to monitor the school's behavior intervention program for any student being

considered for or receiving aversive behavioral interventions to ensure the protection of legal and human rights of individuals.

- (ii) The Human Rights Committee shall be comprised of individuals not employed by the school or agency, which shall include at least one licensed psychologist with appropriate credentials in applied behavior analysis, one licensed physician, physician's assistant or nurse practitioner, one registered dietician or nutritionist, one attorney, law student or paralegal and one parent or parent advocate. In addition, when the purpose of the Human Rights Committee meeting includes a review of an individual New York State student's program, a representative of the school district or agency placing the student in the program and a representative of the Department shall be invited to participate.
- (iii) The Human Rights Committee shall meet at least quarterly to review, monitor and investigate the implementation of students' behavioral intervention plans that include aversive behavioral interventions. A written report on the findings and recommendations of the Human Rights Committee regarding an individual student shall be provided to the CSE or CPSE of the student and to the agency that placed the student in the program.
- (13) Behavioral intervention plans shall be designed and supervised by qualified professionals in accordance with their respective areas of professional competence. All personnel involved in the development, application, monitoring, data collection or review of a behavioral intervention plan that includes the use of aversive behavioral interventions shall be appropriately certified in accordance with the provisions of Part 80 of this Title and sections 200.6 and 200.7 of this Part.
- (14) All staff and volunteers who will be authorized to use aversive behavioral interventions on students shall receive appropriate supervision, including direct observation.
- (15) Appropriate training shall be provided to staff on a regular, but at least annual basis, which shall include, but not be limited to, training on:

- (i) safe and therapeutic emergency physical restraint interventions;
 - (ii) data collection of the frequency, duration and latency of behaviors;
 - (iii) identification of antecedent behaviors and reinforcing consequences of the behavior;
 - (iv) approaches to teach alternative skills or behaviors including functional communication training;
 - (v) assessment of student preferences for reinforcement;
 - (vi) assessing and responding to the collateral effects of the use of aversive behavioral interventions including, but not limited to, effects on a student's health, increases in aggression, increases in escape behaviors and/or emotional reactions;
 - (vii) privacy rights of students; and
 - (viii) critical incident reporting.
- (16) Aversive behavioral interventions shall only be provided with the informed written consent of the parent and no parent shall be required by the program to remove the student from the program if he or she refuses consent for an aversive behavioral intervention.
- (17) The program's use of aversive behavioral interventions, including a review of all critical incident reports relating to such interventions, shall be subject to quality assurance reviews to ensure that practices are clinically sound, supported by proper documentation and consistent with these program standards and the school's policies and procedures as approved by the Department.
- (18) The program shall submit quarterly written progress reports on the implementation of the student's behavioral intervention program to the CSE or CPSE and to the agency that placed the student in the program.
- (19) A school district that places a student in a program that uses aversive behavioral interventions with such student shall be responsible to ensure that the student's education and behavior program are being implemented consistent with the recommendation of the CSE or CPSE as documented in the student's IEP and behavioral intervention plan, which shall include the review of written progress monitoring and critical incident reports, at

least annual observations of and, as appropriate, interviews with the student in the program and regular communication with the student's parent. The CSE or CPSE shall convene at least every six months, or more frequently as needed, to review the student's educational program and placement for any student for whom the CSE or CPSE has recommended the use of aversive behavioral interventions.

- (20) The use of aversive behavioral interventions in education programs operated pursuant to section 112 of the Education Law and Part 116 of this Title are subject to the rules of the respective State agency operating such programs where such rules are inconsistent with the requirements in this section.

EXHIBIT 37

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UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF NEW YORK

JEANETTE ALLEYNE, ET AL.

Plaintiffs,

v.

Civil No. 1:06cv00994
(GLS)

NEW YORK DEPARTMENT of
EDUCATION, ET AL.

Defendants.

APPEARANCES:

OF COUNSEL:

FOR PLAINTIFFS:

O'Connell, Aronowitz Law Firm

JEFFREY J. SHERRIN, ESQ.

MEMORANDUM-DECISION AND ORDER

On August 16, 2006, The Judge Rotenberg Educational Center, Inc. ("JRC") and the parent(s) and/or guardians of identified individual students filed suit against the New York State Education Department, its Commissioner, Richard P. Mills, and the New York State Board of Regents ("State Education Defendants") alleging, *inter alia*, violations of the Individuals with Disabilities Education Act ("IDEA"), 20 U.S.C. §§ 1400

et seq. The gravamen of the complaint is that the State Education Defendants unilaterally altered the students' individualized educational programs ("IEP's") when they passed June 20, 2006, Emergency Regulations that eliminated or restricted aversive treatment that had been authorized for the individually named students by their parents or guardians, their IEP's, and orders of a Massachusetts probate court. The complaint was accompanied by an application entitled, "TRO/Preliminary Injunction," which, *inter alia*, sought to preclude the State Education Defendants from enforcing the Emergency Regulations.

Because the plaintiffs failed to submit an affidavit demonstrating the need for the court to consider their application *ex parte*, the court declined to do so. Instead, the court ordered expedited service of plaintiffs' papers, and scheduled a hearing for August 22. The State Defendants subsequently filed an expedited response, and the court conducted the hearing. As prompted by the court, plaintiffs subsequently filed an amended complaint on September 1 which added new, individual plaintiffs.

As the court indicated during the hearing, it is fully conversant with the legal standard applicable to equitable relief sought by a temporary restraining order or preliminary injunction. To warrant a temporary

restraining order, a plaintiff must satisfy the same prerequisites as a party seeking a preliminary injunction. *Local 1814, Intern. Longshoremen's Ass'n, AFL-CIO v. New York Shipping Ass'n, Inc.* 965 F.2d 1224, 1228 (2d Cir. 1992). In general, a district court may grant a preliminary injunction where the moving party establishes: (1) that it is likely to suffer irreparable injury if the injunction is not granted, and (2) either (a) a likelihood of success on the merits of its claim, or (b) the existence of serious questions going to the merits of its claim and a balance of the hardships tipping decidedly in its favor. *Moore v. Consolidated Edison Co. of New York, Inc.*, 409 F.3d 506, 510 -511 (2d Cir. 2005). "Such relief...is an extraordinary and drastic remedy, one that should not be granted unless the movant, by a clear showing, carries the burden of persuasion." *Id.* However, when the moving party seeks to stay governmental action taken in the public interest pursuant to a statutory or regulatory scheme, the injunction should be granted only if the moving party meets the more rigorous likelihood-of-success standard. *Connecticut Dept. of Environmental Protection v. O.S.H.A.*, 356 F.3d 226, 230 -231 (2d Cir. 2004)(quoting *Beal v. Stern*, 184 F.3d 117, 122 (2d Cir.1999) (citations and internal quotation marks omitted)). Moreover, in some cases, a significantly higher standard

applies. The moving party must make a "clear" or "substantial" showing of a likelihood of success in two instances; namely, (1) the injunction sought is mandatory, *i.e.*, "will alter, rather than maintain, the status quo"; or (2) the injunction sought "will provide the movant with substantially all the relief sought, and that relief cannot be undone even if the defendant prevails at a trial on the merits." *Jolly v. Coughlin*, 76 F.3d 468, 473 (2d Cir.1996).

During the hearing, the court attempted to share its equitable concerns given the arguments raised by the parties. Without reciting each of those argument and without reaching any legal conclusions, the court repeats several of its concerns. The court is not convinced that it has a jurisdictional basis to grant preliminary relief to those who are not parties to this litigation. If the court does grant preliminary relief, the language employed should be narrowly tailored to encompass only those named plaintiffs attending the JRC whose *status quo* was altered by the Emergency Regulations and who now express the definitive wish to return to the *status quo ante*. The court is mindful that the State Educational Defendants are authorized by the IDEA to set standards governing special education and related services. While the plaintiffs must make a "clear" or "substantial" showing of a likelihood of success if a preliminary injunction

"will alter, rather than maintain, the *status quo*", it is the State Education Defendants that altered the *status quo* when it passed the Emergency Regulations. Furthermore, the court believes that narrowly tailored relief can be undone if the State Education Defendants prevail on the merits, and the court has the means to expedite disposition.

Ultimately, the court is confronted with a dispute concerning the efficacy of aversives in terms of what, when and how they should be utilized, and whether the State authority to regulate them has been exceeded. Broadly speaking, the State and JRC are on opposite sides of the debate. However, it is obvious that at least some parents have shouldered the unenviable task of caring for severely challenged children for years, believe in the efficacy of aversives as applied to their children, and are now caught in the middle. Accordingly, the court clearly intended to convey to the parties that it would look favorably on the *status quo ante*, expedite the action, and grant a narrowly tailored preliminary injunction.¹

Based upon these equitable observations, the court requested that the parties confer and provide a proposed order by September 1, narrowly

¹See by analogy, 20 U.S.C. § 1415(j) (IDEA stay-put provision).

tailored to address the court's concerns. While plaintiffs provided a proposed order, it far exceeded the narrow scope intended by the court. While the State Education Defendants continue to object to any preliminary relief, they have provided suggested limits that would effectuate the court's concerns.

Therefore, and for the reasons stated, it is hereby

ORDERED that the State Education Defendants are preliminarily enjoined from the enforcement of 8 NYCRR §200.22(f)(2)(vi) (limiting the use of aversives to aggressive and self injurious behavior) and 8 NYCRR § 200.22(f)(2)(ix) (prohibiting the combined use of aversive interventions with mechanical restraints), **only as** those named Student Plaintiffs identified in the September 1, 2006, amended complaint who: (1) have a current individualized education program (IEP) that expressly permits Level III aversives, and that permitted Level III aversives on June 23, 2006; (2) have a current behavioral intervention plan that specifies the aversive intervention appropriate for each targeted behavior; and (3) have a current Massachusetts Probate Court order that authorizes the use of Level III aversives, and had such an order in effect on June 23, 2006; and it is further

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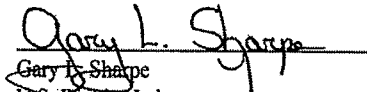
ORDERED that to the extent plaintiffs' proposed order seeks broader relief, it is **DENIED**; and it is further

ORDERED that in the interest of expedience, this court's order of referral to Magistrate Judge Treece is rescinded, this court will preside over the pretrial management phases of this litigation, and a Rule 16 conference is set for October 19, 2006 at 1:30 P.M.; and it is further

ORDERED that the parties **shall** file a joint Rule 16 Management Plan on or before October 12, 2006.

SO ORDERED.

Date: September 8, 2006
Albany, New York



Gary L. Sharpe
U.S. District Judge

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**UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF NEW YORK**

JEANETTE ALLEYNE, ET AL.

Plaintiffs,

v.

**Civil No. 1:06cv00994
(GLS)**

**NEW YORK DEPARTMENT of
EDUCATION, ET AL.**

Defendants.

**APPEARANCES:
FOR PLAINTIFFS:**

OF COUNSEL:

Students

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**JEFFREY J. SHERRIN, ESQ.
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JRC

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Albany Office
The Capitol
Albany, NY 12224

KELLY MUNKWITZ, ESQ.
Assistant Attorney General

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It is hereby

ORDERED that the Court's preliminary injunction dated September 8, 2006 shall also be deemed to apply to those named Student Plaintiffs as identified in the September 1, 2006 amended complaint or any amended complaint subsequently filed with leave of the Court who: (1) had an individualized educational program (IEP) that expressly permitted Level III aversives on June 23, 2006; (2) have or will have a current behavioral intervention plan that specifies the aversive intervention appropriate for each targeted behavior; (3) have or will have a Massachusetts Probate Court order that authorizes the use of Level III aversives; and (4) whose parent and/or legal guardian continues to consent to the use of Level III aversives as described in his/her child's IEP in effect on June 23, 2006; and it is further

ORDERED that the State Education Defendants are preliminarily enjoined from the enforcement of 8 N.Y.C.R.R. § 200.22(e)(1)(ii) (requiring the submission of an application to the Commissioner (the "Application") concerning the use of aversive behavioral interventions by October 1, 2006) only as to those named Student Plaintiffs described above whose school district or committee on special education (CSE) removes or have removed Level III aversives from their IEP after June 23, 2006 and whose

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parent and/or legal guardian files a request for an impartial hearing with the school district or the CSE within thirty days of the written notification to the parent or guardian that level three aversives were removed from the IEP, for so long as the proceedings under Section 1415 of the Individuals with Disabilities Education Act (IDEA), 20 U.S.C. § 1400 et. seq., including judicial appeals, are pending; and it is further

ORDERED that the State Education Defendants are preliminarily enjoined from the enforcement of 8 N.Y.C.R.R. § 200.22(e)(1)(ii) only as to those Student Plaintiffs described above whose school district or CSE has not removed Level III aversives from the child's IEP but for whom an Application has not been filed; and is further

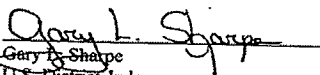
ORDERED that the Plaintiffs are granted leave to amend the complaint, in compliance with Rule 15, to add parties who meet the above criteria; and it is further

ORDERED that this Order shall remain in force and effect until further order from this Court, and it is further

ORDERED that the Clerk shall provide a copy of this Order to the parties.

IT IS SO ORDERED.

Dated: October 2, 2006.
Albany, New York


Gary L. Sharpe
U.S. District Judge